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PRESIDENTIAL ADDRESS

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Santa Fe, New Mexico

(Delivered before the New Mexico Medical Society, at its Forty-Eighth Annual Meeting, Raton, N. M., June 4-6, 1930.)

First, let me express my appreciation of the honor done me in my election as President of the New Mexico Medical Society for 1930-31. I know that doctors as a whole are not given to praising themselves, but I should like to register my opinion, which has been strengthened year by year, that the doctors of New Mexico are a group of men meriting and receiving great respect as individuals in human society, and, as a group amongst the larger group of physicians and surgeons of the United States, for their professional ability. The compliment of being elected the president of a society made up of such people is, to my mind, a real one.

For this compliment I thank you, and pledge you my sincere efforts to be of some service to you, and to your efforts for others. The routine work of the Society I shall do my best to take care of properly, humbly trying to learn at the feet of Doctor Cohenour and others what I do not know.

I think we all realize that we suffer at times from a lack of cohesion in the state society, and in the foundation of the state society, the county medical societies. Distances are great and the population widely scattered in New Mexico, but I think that we all agree that the local gatherings of the physicians, which these distances make difficult, are very much worth while. Whether the main feature of the meeting of the county society be a formal paper, or informal discussion, clinical reports, discussion of case reports, as those of the Massachusett General Hospital, or chiefly, or even purely, social gatherings, the practicing physician's mind is cleared, and his proficiency increased by contacts with his professional brothers and their ideas, and, perhaps as important as anything else, it affords an opportunity to get straightened out on the small or large misunderstandings that always have and always will come up between human beings whose orbits of activity meet and overlap. Perhaps your officers, councillors and delegates can help in the full functioning of the county societies; it is worth trying, and I feel sure that the basis of a coherent and cohesive state society is the actively functioning county medical society.

Only by a coherent and cohesive state society can we secure for ourselves, our profession and for society at large in New Mexico the things which we desire. These desires fall naturally in certain divisions; our own legitimate economic desires; our desires to improve ourselves professionally; and our desire to render the greatest service possible to that society of which we are members. Efforts properly balanced toward furthering all these desires further each other and, in the end, most largely, the third—that for service to our communities. After all, I believe that in gratifying this last desire, we secure our greatest satisfaction and our fullest repayment.

There are numerous ways in which the medical profession, as a group, can render service to the community, besides the daily practice of their profession. Among these are, encouragement of the work of the Bureau of Public Health, the value of whose work, because of our specialized education, we appreciate more than the layman; aid and encouragement of the work of the State Bureau of Child Welfare, and of the various local units of welfare work, private, municipal, county, district and state, which help to fill in the gaps between what the patient

can afford to have and what they should, or must have; education of legislators amongst our patients to the needs of these Bureaus, financial and legislative; and, last, but certainly not least, education of our legislators to the need for adequate protection for the layman citizen of the state from the practitioner of the healing arts whom we know not to be adequately educated in these arts.

It seems rather ridiculous to us, who have spent many years and much hard labor in acquiring knowledge fitting us to practice the healing art, that the layman is not more astute in his appreciation of what knowledge is necessary thereto. We know what a highly intricate and complicated machine the human body is. The layman, having usually absolutely no education in anatomy, physiology and the like, does not. But he does have a child like faith in letters of any kind, before or after a name, and especially the title "Doctor." He has not insisted that the title legally, shall mean much, but he believes it does. And he expects great knowledge from the man who sports the title, in which he is often greatly deceived. Does not our greater knowledge, a knowledge which we have been enabled to secure through the organization by society of colleges and universities, place an obligation on us to protect the less educated of society from the uneducated and unqualified practitioner of the healing arts?

All civilized countries have made attempts to protect its citizens in some manner, by medical practice acts which are variously successful. In the United States and in New Mexico particularly, the physician of the regular school has cleaned his house well and has it pretty well in order. We have in New Mexico as admirable a set of regulations governing the practice of medicine by the physician of the regular school as could be desired, and a Board of Medical Examiners whose work is above reproach. But our medical practice acts, as regards other than the regular school, are very poor. Most of us here are at least fairly well acquainted with the efforts made in the past to rectify these shortcomings. Some of us are painfully well acquainted with them. The last attempt was made three years ago in the legislature, and failed through lack of preparatory work on the leislators, a lack of interest on the part of the physicians, which I believe was more apparent than real, and a far too active interest in opposition to the bill on the part of some of the practitioners of the healing arts other than the regular school.

The suggested act was one for a combined board—a not entirely satisfactory type at best, and perhaps we are as well off without it. It suffered from a defect rather common in bills controlling the practice of the healing arts; it took care of present evils fairly well, but made no provision for new schools of medicine, at present either just arising or non-existent. A medical practice act should do both, to be efficient or just. And a good medical practice act must avoid direct frontal attack, for the various schools of medicine are too deeply intrenched, with much financial outlay for colleges, equipment, propaganda and the like, as well as many individuals engaged in practice, to be dislodged without great difficulty by such an attack. Nor is amalgamation of the present various schools of medicine advisable, with the idea of starting over again with a clean sheet; this solves nothing and makes no provision for new schools of practice.

There is one type of control of the practice of the healing arts which seems to fit the situation we have here in New Mexico, with our separate licensing boards, better than anything else. This is the so-called basic science act. Such acts are already in force in six states: Connecticut, Minnesota, Nebraska, Washington, Wisconsin, Arkansas and in the District of Columbia, in all of which during the years they have been in force, from 1927 on, have proved fairly satisfactory. They are eminently fair and reasonable, to such a degree indeed that their reasonableness is at once evident even to a distracted legislator in the heat of a campaign or a legislative session. They provide for present established schools of practice as well as for future ones. And they provide a fair degree of protection for the layman uneducated in the details of his own make-up.

The basic science acts recognize the existence of various schools of medical practice, but also recognize the basic sciences, which must underly all practice of the healing arts, such as anatomy, physiology, chemistry, bacteriology and pathology. These sciences are not matters of dogma, but of observed facts. By seeing to it that all practitioners of the healing arts know something of these basic sciences, no matter what their school of treatment, society is protected to a certain extent. To avoid prejudice, it is provided that the examiners shall not be in the active practice of any school of the healing arts, and that all practitioners, of whatever school, shall take the examination. They meet the tangle as to requirements in preliminary education by allowing even the high school graduate to take the examination. On presentation of a certificate granted on passing the examination in the basic

sciences, the various licensing boards for the various schools of medicine may make further examination in the same subjects, or in other sujects only, as they desire, and they may further make their own requirements for years of study, preliminary or medical, hospital service, and the like. They do not, therefore, pull all the schools down to a common low level but rather permit each to rise to as high a level as it may wish, at the same time providing a minimum below which they may not fall. Reciprocity is provided for states with basic science acts, or conducting actual examination in the same sciences. They do not restrict the patient to a single choice as to the method of diagnosis and treatment but do provide that he shall be at least partially protected against unqualified practitioners in whatever method he elects.

It seems to me that New Mexico badly needs some added protection for the medically uneducated layman; that logically the state might look to the organized medical profession to suggest a method for securing such protection; that in suggesting it we are furthering our own legitimate, selfish, as well as unselfish, desires; and that the so-called basic science acts offer the best method of securing such protection to our fellow citizens. I have, therefore, brought it to your attention here, and would ask you to consider it, with the possible idea of some recommendation to your officers, council and house of delegates as to action which you may desire.

THE HEART IN TUBERCULOSIS, WITH PRELIMINARY REPORT OF ELECTROCARDIOGRAPHIC STUDY OF 105 CASES

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(Read before the Arizona State Medical Association, April 24 to 27, 1930, at Phoenix, Arizona, and published wth the permission of the Medical Director of U. S. Veterans' Bureau, who assumes no responsibility for opinions expressed or conclusions drawn by the writer.)

A properly functioning heart, one that does not draw too heavily upon its own reserve power, is one of the most valuable assets of the tuberculous patient. That there exists a very definite interrelation, interdependence and association between the cardiovascular and respiratory systems is an accepted fact. Any impediment, either within the heart or along the course of the greater or lesser circulation, which interferes with the normal propulsion of the blood stream, naturally increases the heart load. This increased load continued over a long period of time, or an unusually heavy load for a shorter period, will utilize the reserve force and eventually result in a temporary or permanent weakening of the myocardium.

Dr. Lemon[1], of the Mayo Clinic, in a recent study, "The Effect of Chronic Pulmonary Disease on the Volume and Composition of the Blood," has demonstrated that an impaired function of the heart muscle results from the increased load placed upon the organ, due to disturbance in the pulmonary circulation in certain pathologic conditions in the thorax.

A discussion by Dr. Rappoport[2], of Denver, on "Anoxemic Syndrome as a Cause of Myocardial Insufficiency in Pulmonary Tuberculosis," appears logical and conclusive.

As clinicians, we frequently observe a symptomatology, especially in pulmonary tuberculous patients, which under ordinary circumstances would direct our attention to the heart as a causative factor. These symptoms, I fear, are too frequently disregarded, simply "passed over," as a part of the pulmonary syndrome, the stethoscope probably failing to reveal sufficient evidence to warrant a diagnosis of "heart disease." We cannot rely too much upon positive physical findings. The "silent heart," the heart in which no adventitious sounds are heard, may be the most dangerous type. The myocardial insufficiency associated with and resulting from the chronic pulmonary tuberculous lesion, is frequently overlooked because of the absence of outstanding objective and subjective symptoms. Faint heart sounds, possibly diminished pulse pressure, occasionally a systolic murmur heard over precordium, being the only positive objective findings. The subjective symptoms may be negligible so long as patient remains at rest. Palpitation, precordial pain, arrhythmia, tachycardia, cyanosis, and dyspnea following exercise or the taking of a heavy meal, are significant.

In considering myocardial insufficiency, it is important that we think of the five automatic qualities of the heart muscle, namely: tonicity, contractility, conductivity, irritability and rythmicity. In any long-continued toxic state, such as is observed in tuberculosis, we may find any one or all of these properties disturbed.

Tonicity of the heart muscle is that quality which resists over-distention in diastole and guards against the loss of a "safe footing" for the recurring active contraction. Ionus deficiency is probably the basic prin-

ciple of myocardial insufficiency of which we are talking. There may be present a pre-existing myocardial abnormality resulting from some previous infectious process. Evidence of myocardial degeneration may be found in the heart so affected.

Certain variations are observed in the electrocardiogram, in a large percentage of tuberculous cases, presenting symptoms suggestive of cardiac dysfunction. The significance of these changes is not understood as yet: however, the frequency of a low P wave, certain irregularities of the Q.R.S. complex, a flattening of the T wave, are, we believe, indicative of myocardial hypotonicity.

Disturbance in conduction as shown by prolongation of the P. R. interval, complete block, widening and other abnormalities of the Q.R.S., as found in bundle branch block and certain irregularities of the Q.R.S. indicating arborization block, and increased irritability as manifested by premature systoles, fibrillation and paroxsymal tachycardia, have been observed with varying degrees of frequency. Cases have not been studied in sufficient number in this series to justify a conclusion as to the comparative prevalence of these abnormalities..

Reserve is that quality which gives elasticity to the heart and provides it with power to maintain a normal output and meet the demands of the body under extremities, represented by absolute body rest on the one hand, and the most violent exertion on the other. This surplus power is a variable factor, lower in the asthenic individual with a small drop-type heart, and is a force most difficult to evaluate because of our inability to measure damage done to the myocardium by some previous infectious or toxic process. It is the degree of impairment or exhaustion of reserve force, the sudden or gradual narrowing of the field of response, which determines cardiac insufficiency. Our prime objective, then, should be the early recognition of this disturbance in function and, so far as possiole, to measure the degree of impairment while there is an opportunity to amplify, restore or rehabilitate myocardial reserve.

The pathologist is unable to demonstrate at necropsy a concrete change in the myocardium which fully explains the clinical picture of the failing heart. Myocardial insufficiency, as we observe it in the tuberculous patient, is similar to the insufficiency found in other toxemic states. It is not a myocarditis. It may or may not have left evidence of a degenerative change in the muscle cells. Dr. Lyman Green[3] states that "a heart which certainly killed its possessor is not of necessity the seat of such demonstrable, specific and concrete changes as are adequate to explain death." Dr. Robertson[4], pathologist of the Mayo Clinic, and Dr. Henry Hartman[5], former professor of pathology, University of Texas, state that macroscopical findings at necropsy justify the clinical diagnosis of insufficiency. However, there are no specific microscopical changes found. Dr. Hartman further expresses the opinion that hypertrophy cannot exist in the active toxic tuberculous patient, because of the inability of overworked muscle to retain sufficient nutrition. Hypertrophy, however, is not uncommon where there exists moderate to extensive pulmonary fibrosis, in a non-toxic, well-nourished patient. Dr. Blumberg, pathologist at one of the larger Veterans' Bureau hospitals, explains myocardial failure in tuberculosis as follows: "The increased work of the heart; the failure to furnish sugar to muscle tissue, as result of toxic state, associated with fever and the lessened exogenous supply, deprives the muscle tissue of the necessary sugar content. These factors, in addition to the formation of fatigue bodies, as well as sarcolactic acid, decrease the power of the myocardium to the point where its working capacity will fail."

In our series of 105 cases of pulmonary tuberculosis studied at this hospital, practically all presented some symptoms suggesting heart dysfunction. An attempt has been made, by means of careful consideration of history, the subjective symptoms, physical findings, and electrocardiographic changes, to evaluate the extent of impairment. In many instances, additional information was supplied through laboratory tests, such as study of twenty-four hour specimen of urine, blood chemistry, basal metabolism and vital capacity.

Approximately seventy per cent of the series of 105 cases were classed as having myocardial insufficiency of minimal, moderate or severe degree. Five case histories representing patients who gave positive clinical history, physical findings, or electrocardiographic changes, are submitted.

Table No. 1 gives tthe average loss of weight and average systolic and diastolic blood pressure in the 105 cases.

Table No. 2 tabulates the abnormalities noted in the cardiograms.

(Table No. 1)

Average loss of weight			Average Blood Pressure			
Classification	No.	Pounds	Classif'c'tn	No.	S	D
C	45	36	C	45	107	74
B	43	21	B	43	115	78½
A	13	5½	A	13	120	79
Inactive	4	0	Inactive	4	115	76

ELECTROCARDIOGRAPHIC FINDINGS

(Table No. 2).

Inverted R, Lead I (Incicating right axis deviation)			S predominating, Lead III (Indicating left axis deviation)		
C	21		C	8	
B	9		B	5	
A	3		A	2	Total 15
Inactive	1	Total 34			

P below 1 mm., Main Lead			P notched in Main Lead		
C	26		C	14	
B	21		B	11	
A	3		A	4	Total 29
Inactive	3	Total 53			

QRS Complex below 7 mm., Main Lead			T below 2 mm., Main Lead		
Classification	No	Total	Classification	No	Total
C	11		C	26	
B	6		B	14	
A	3		A	2	
Inactive	1	21	Inactive	1	43

RT coming off above Base Line			Convex RT		
C	5		C	4	
B	5		B	7	
A	4	14	A	4	15
Inactive	1	31			

Rate above 100		
C	21	
B	7	
A	2	
Inactvive	1	31

The following cases are presented as representative of the group presenting subjective symptoms indicating varying degrees of myocardial insufficiency.

The electrocardiographic changes, while not definitely diagnostic, in each instance, are, we believe, worth consideration.

CASE 1.

Male, white, age 50. Occupation: assistant general storekeeper.

Family history: Negative.

Personal history: Typhoid at age of 12. Malaria, Spanish-American war. Recurrent attacks of chills and fever until 1920. Pneumonia also during Spanish-American war. Icterus in 1919, duration 15 months. Arthritis, 1916-17. Diagnosed pulmonary tuberculosis in 1928. Symptoms for one year prior to diagnosis.

Admitted to this hospital in November, 1929. Diagnosed tuberculosis, pulmonary, chronic, far advanced, "B". Fibrosis right upper, fibro-caseous, inf ltration left upper, cavity one inch in diameter. Subjective symptoms: Cough, loss of weight, weakness, shortness of breath for past two years, more severe past few months. Was told he had "asthma." Dyspnea on slight exertion persists. "Fluttering" in region of left nipple. Attacks of short duration, leave sensation of soreness. Vertigo on rising from prone position.

Heart study December 1, 1929. Blood pressure: systolic, 106; diastolic, 74. Sounds faint, presystolic and systolic murmur precordium.

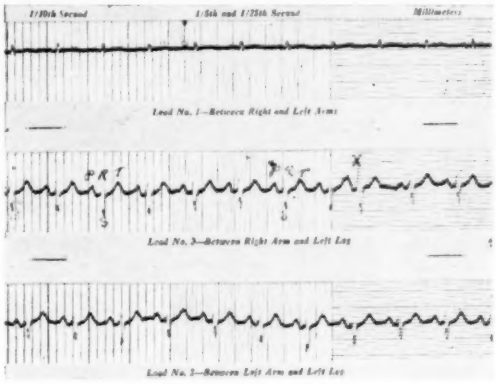

Fig. 1

Urine negative. Wassermann negative. Sputum negative. Cardiogram (Fig. 1) made, showing rate 100 X premature systole. Note approximate equal voltage of R and S in leads 2 and 3; S notched in leads 2 and 3. Dec. 1, 1929. Treatment: rest, low-protein diet. Digitalized and maintained 3 months.

March 21, 1930: Recheck cardiogram made, showing rate 86, with no appreciable change in deflections. Urine: Specific gravity, 1.026; albumin, negative; occasional WBC; total N., 14.6; urea, 31.2; creatinin, 0.7; uric acid, 0.7; chlorides, 14. Blood chemistry: NPN, 23.1; urea, N 9.4; creatinin, 1.4; uric acid, 1.3; sugar, 112.

General condition improved. Practically no shortness of breath so long as digitalis is continued.

Diagnosis: Myocardial insufficiency.

CASE 2.

Male; white; age 30; occupation, miner.

Family history: Father died of heart trouble; one sister died of tuberculosis.

Personal history: Tonsillectomy, 1923. Metal mining for five years, intermittently, prewar. Discharged, December, 1918, no disability. Returned to work, developed "colds," cough. Diagnosed bronchitis but had streaked sputum. Continued work

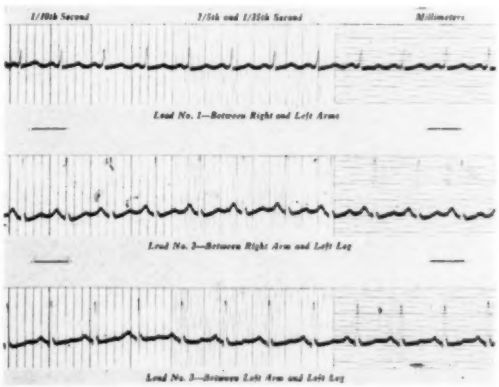

Fig. 2

at intervals. Pulmonary hemorrhage, 1924. Diagnosed tuberculosis, 1928.

Admitted to this hospital February, 1930. Diagnosed tuberculosis, far advanced "B." Fibrocaseous infiltration middle and upper lobes, right, fibrocaseous infiltration both lobes left; cavity both uppers. Normal weight, 147; admission weight, 135. Blood pressure: systolic, 118; diastolic, 84. Temperature range, 97.8-99. Sputum, positive. Urine, negative. Blood chemistry: NPN, 31.4; urea N, 12.4; creatinin, 1; uric acid 1.2; chlorides, 598; sugar, 88.9; calcium, 10.4. Basal metabolism rate, plus 16.18 per cent. Vital capacity, 2.4 liters. Electrocardiogram (Fig. 2) made March 11, showing rate 112, sinus rhythm; flat T wave and slight downward convexity of R-T wave. Treatment, bed rest, regular diet. Digitalized and maintneance since March 13. Cardiogram made April 10, condition improved. Diagnosis: Myocardial insufficiency.

CASE 3.

Male: white; age 34; occupation, salesman.

Family history: Negative for tuberculosis and heart disease.

Personal history: "Membraneous croup" in early childhood. Influenza six or seven years ago. Symptoms suggestive of pulmonary tuberculosis in 1921; noghtsweats, fatigability, cough, loss of weight, positive sputum, 1924. Positive x-ray, 1926. Worked until November, 1926. Hospitalized in 1927, aphonia. No improvement. Edema of face and extremities, three months prior to admission to this hospital. Periodic diarrhea.

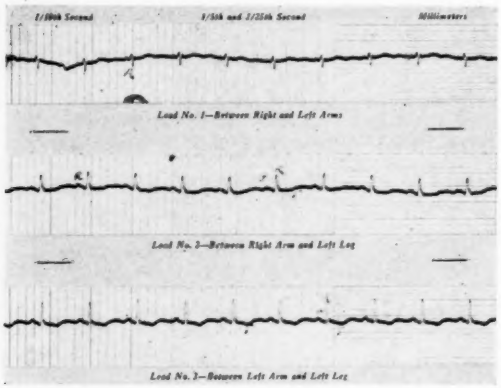
1/10th Second
1/5th and 1/25th Second
Millimeters
Lead No. 1—Between Right and Left Arms
Lead No. 2—Between Right Arm and Left Leg
Lead No. 3—Between Left Arm and Left Leg

Fig. 3

Admitted to this hospital, November 26, 1929. Diagnosed tuberculosis pulmonary, far advanced "C." X-ray indicated far advanced tuberculosis left, marked lung contraction and large cavity, moderately-advanced fibrotic right. Displaced heart. Laryngitis, tuberculous. Heart study December 1, cardiogram made (Fig. 3). Rate 100, sinus rhythm; inverted R lead 1; low voltage of all deflections; flat T wave in leads 2 and 3 and inverted in lead 3; low P wave in all leads. Sedimentation test: index, 33 mm., markedly active. Urine: specific gravity, 1.008; albumin, 0.1 per cent; occasional fine granular cast. Blood chemistry: NPN, 31.5; urea N, 12.4; creatiniu, 1.2; uric acid, 1.6; chlorides, 412; sugar, 12.5. RBC: 3,400,000. Leukocytes: 19,200. PM, 85. Hemoglobin 70 per cent.

Symptoms: Edema, general, marked extremities and genitals. Periodic diarrhea. No marked respiratory embarrassment. Urine output much below normal. Temperature range, 36-37.8; pulse, 80-100 on admission. Sputum negative for tuberculosis. Wassermann negative. Treatment: rest, low-liquid, low-protein diet. Digitalized. Ammonium chlorid, 60 gr. daily; novasurol intravenously, daily, beginning with 1 c.c., increased to 2 c.c., continued five to six doses. Intake, 4th day, 1300 c.c.; output, 1800 c.c. 6th day; intake, 1450 c.c.; output, 2600 c.c. Edema gradually subsided. General condition improved. Maintenance does digitalis continued to date.

January 3, 1930: Temperature, 36-37. Pulse, 72-90. Blood chemistry: NPN, 32.3; urea N, 12; creatinin, 1; uric acid, 1.4; chloride, 460; sugar, 111.

February 13, 1939: Urine: specific gravity, 1.010; albumin, 0.02 per cent; numerous fine granular casts.

February 13, 1930: Urine: specific gravity, 1.010; improvement in T wave. Pulse and temperature unchanged. Urine same.

March 21, 1930: 24 hour specimen urine: 1400 c.c; reaction, acid; specific gravity, 1.015; albumin, trace; occasional WBC; fine granular casts; moderate amount. Total N, 21.2; urea, 41.6; creatinin, 0.8; uric acid, 0.7; chlorides, 14. Blood chemistry: NPN, 40; urea N, 16.6; creatinin, 1.2; uric acid, 1.62; chlorides, 466.4; sugar, 80. Blood: RBC, 3,300,000; leukocytes, 11,800; PM, 75; hemoglobin, 70 per cent. Basal metabolism rate, plus 0.43 per cent. Vital capacity, 1.4 liters. No edema, patient comfortable, up and around ward.

Diagnosis: Myocardial insufficiency; nephritis, chronic, interstitial.

CASE 4.

Male: white; age, 30; occupation, real estate and insurance agent.

Family history: Father died of apoplexy.

Personal history: No history of infectious disease other than influenza while in service. Discharged, December, 1918: no disability noted. Worked as bank clerk. In 1920, began losing weight, pulse rate increased. Was told that he had high blood pressure. Continued to work, but symptoms increased. Diagnosed pulmonary tuberculosis in 1927. Sputum positive.

Admitted to this hospital, June, 1929. Diagnosed tuberculosis, far advanced "C." Fibrosis right upper. Almost complete destruction of left lung. Temperature range, 36.6-38, gradually receding to normal range within two weeks; pulse, 110-130; usual after first week, 80-120. Normal weight, 175; present weight, 107. Blood pressure: systolic, 98; diastolic, 70. Urine: specific gravity, 1.030; albumin, .2 per cent; numerous fine granular and hyaline casts. Sputum, positive. Cardiogram (Fig. 4) made

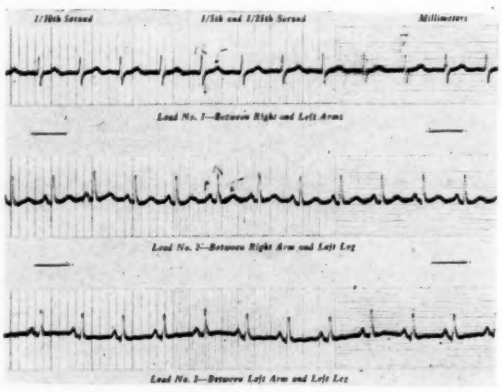
1/10th Second
1/5th and 1/25th Second
Millimeters
Lead No. 1—Between Right and Left Arms
Lead No. 2—Between Right Arm and Left Leg
Lead No. 3—Between Left Arm and Left Leg

Fig. 4

June 14, 1929: rate 120, sinus rhythm; low or absent P in lead 1, low T in leads 1 and 2 and isolectric lead 3; R and S practically equal in lead 1. Blood pressure: systolic, 110; diastolic, 80. Treatment: rest,

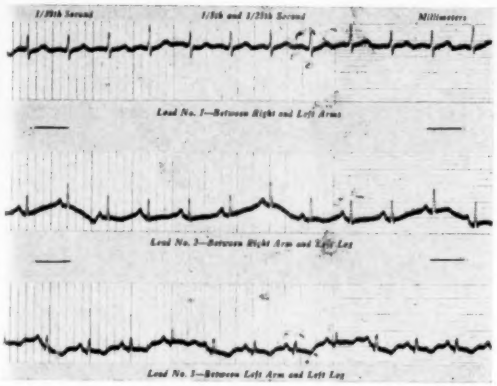
Millimeters
Lead No. 1—Between Right and Left Arms
Lead No. 2—Between Right Arm and Left Leg
Lead No. 3—Between Left Arm and Left Leg

Fig. 5

symptomatic. Condition became weaker. Urine same. Temperature within normal range. Pulse, 110-120. September 29 complained of dropped heart beats and dyspnea. Cardiogram, rate 120, T wave slightly flatter in leads 1 and 2. Patient slightly cyanotic. Systolic murmur at apex. Second pulmonic accentuated. Soon after this date patient began to show signs of edema of lower extremities. Dyspnea and cyanosis more marked until November 24, when patient was digitalized and kept on maintenance dosage for six weeks. Edema increased. Dyspnea more marked. Ammonium chloride and novasurol administered. Fluid limited; poor response on account of patient's failure to cooperate. Forced to cease efforts to eliminate. Symptoms increased in severity. Cardiogram (Fig. 5) made December 29: rate 120, isolectric T in lead 2, inverted lead 3, voltage of all deflections lower. Patient approaching death. marked edema, dyspnea, cynosis, etc. Patient died, January 3, 1930. No autopsy. Heart death.

Diagnosis: Myocardial insufficiency.

CASE 5.

Male; white; age, 36; occupation, clerk.

Family history: Negative for pulmonary tuberculosis or heart disease.

Personal history: No infectious disease except for pneumonia in service. Diagnosed tuberculosis in 1919. Under medical supervision at home and in hospital since that date.

Admitted to this hospital, November, 1928. Diagnosed tuberculosis, far advanced, active "B"; large cavity right upper, remainder of lung opaque. Normal weight, 150; weight on admission, 136. Blood pressure: systolic, 106; diastolic, 78. Temperature range, 36-37.2; pulse, 62-110. Urine, negative; Wassermann, negative. RBC: 4,000,000; leukocytes, 11,400; PM, 77 per cent. Sputum, positive.

Clinical Course: Ambulant type. Developed symptoms of toxemia, rise in temperature and pulse, March, 1929; symptoms persisted to about May first. Phrenic nerve evulsion, left, May 31, 1929. Normal temperature resumed and continued. Rise of diaphragm could not be detected. Patient had frequent passes; continued to feel good until middle of January, 1930, when he began to complain of digestive disturbances. No serious symptoms until March 1, when he began vomiting, also had diarrhea. Urine: 0.10 per cent albumin; few granular casts. Sputum, negative. Developed edema of lower extremities about March 15, 1930. Cardiogram (Fig. 6) made. Rate 98, sinus rhythm; low voltage all deflections except P; isolectric T lead 1, inverted leads 2 and 3; P-R interval, .16 second. Diagnosed myocardial insufficiency. Blood chemistry—NPN, 39.9; urea N. 15.2; creatinin, 1.6; sugar, 117. Blood pressure: systolic, 100; diastolic, 80. Digital zed, March 19, 1930. Marked anastrca below waist-line, including genitals.

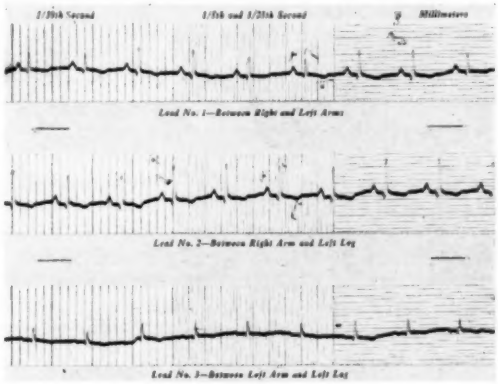
Millimeters
Lead No. 1—Between Right and Left Arms
Lead No. 2—Between Right Arm and Left Leg
Lead No. 3—Between Left Arm and Left Leg

Fig. 6

Treatment: Digitalized. Ammonium chloride, 60 grs., continued 15 to 20 days, reduced to 30 grs. daily. Novasurol intraveneously, 1.2 c.c., March 19 and 20; 2 c.c. March 21, 22, 25, 28 and 31; April 2, 4, 6, 8, and 10, 1.2 c.c.; on April 12 and 14, repeat. March 21, 1930: intake, 1080 c.c., output, 660 c.c. March 22, 1930: intake, 1260 c.c.; output, 1185 c.c.; March 25, intake 2280; output, 1320. March 27: intake, 1380 c.c.; output, 1590 c.c. March 30: intake, 1290 c.c.; output, 1560 c.c. April 4, intake, 2040 c.c.; output, 1920 c.c. April 7: intake, 1800 c.c.; output, 2460 c.c. April 10: intake, 2700 c.c.; output, 4500 c.c. April 14: intake, 2010 c.c.; output, 120 c.c. April 17: intake, 1350 c.c.; output, 3270 c.c.

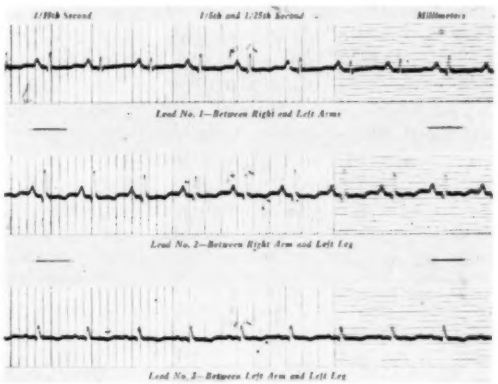
Millimeters
Lead No. 1—Between Right and Left Arms
Lead No. 2—Between Right Arm and Left Leg
Lead No. 3—Between Left Arm and Left Leg

Fig. 7

Free of edema, free of symptoms. Patient sitting up, comfortable. Cardiogram (Fig. 7) made. Rate 90, slight increase in P-R interval; variation in amplitude of R. Condition improved for a time, then relapsed. Blood pressure: systolic, 130; diastolic, 110.

Urine: albumin, 0.25 per cent. April 21, 1930: Urine: albumin trace, few coarse and fine granular casts.

Diagnosis: Myocardial insufficiency; nephritis, chronic, interstitial.

CONCLUSIONS

Any infectious process existing within the physical organism has its effect upon the vital organs. It may be temporary and leave no impairment of function or there may occur a permanent decrease in the organic reserve force.

All normal stimuli are dependent upon a healthy blood stream and the blood itself reacts upon the heart, nourishing the cellular structure in proportion to the essential elements contained therein. In tuberculosis—a chronic infectious disease, releasing to the blood stream daily its quota of toxins, taxing the organs of digestion, in many instances, to the extreme degree, disturbing the metabolic force and upsetting in general the nutritional scheme, destroying the pulmonary tissue and diminishing aeration space—it is but reasonable to assume that permanent damage to the heart will result.

Our experience in hospital service during the past ten years—the clinical observation of a considerable number of cases of pulmonary and other forms of tuberculosis, together with a fairly careful study during the past year (including electrocardiographic records of more than a hundred cases presenting symptoms suggesting cardiac dysfunction) leads us to conclude that diminished cardiac reserve and impairment of tonicity and contractile power, as well as frequent disturbance in other physiological function, is a much more common complication of pulmonary tuberculosis than has generally been recognized. Also, that myocardial insufficiency occurs more frequently among the chronic tuberculous patients than it does in an equal number of non-tuberculous individuals of the same age encountered in the ordinary routine of clinical experience, and that the degree of cardiac impairment is not necessarily in direct proportion to the extent of pulmonary lesion or degree of nutritional disturbance. It is not an uncommon experience to see patients die of tuberculosis, simply waste away, death apparently result of respiratory failure, without showing any clinical evidence of cardiac deficiency. On the other hand, we have often recognized myocardial insufficiency in the well-nourished, ambulent type with a minimal or moderately advanced, possibly inactive, tuberculous lesion. We do not mean to imply, however, that all myocardial insufficiency occurring in patients afflicted with tuberculosis is a direct result of such lesion.

The importance of the cardiogram as a diagnostic aid in myocardial insufficiency, whether it be associated with a tuberculous lesion or not, remains undetermined. It occurs to us that a further observation of the relationship of clinical findings and electrocardiographic changes is worth while. If the electrocardiogram does nothing more than to stimulate more careful observation of heart function in tuberculosis, it will have served a useful purpose.

The failing heart associated with tuberculosis is entitled to application of the same therapeutic procedure as that given similar condition found in non-tuberculous cases. The response of therapy may not be so gratifying but, in many instances, we should be able to bring about a more comfortable state and even prolongation of life.

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ARTHRITIS IN RELATION TO INDUSTRY

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(Read in the Symposium on Industrial Surgery and Medicine, at the thirty-ninth annual meeting of the Arizona State Medical Association, held in Phoenix, April 24 to 26, 1930).

A considerable literature has been accumulating during the last few years concerning the pathology existing in cases of injury to the spine and to the larger joints, in which the patient complains over a longer period of time than is usually required for recovery in similar cases. As x-ray technic improved, changes in the bones and joints were demonstrated which formerly were not suspected, and as these changes were manifestly acquired over a period of time, the question of the responsibility of the trauma for the condition became acutely controversial in courts and before industrial commissions. As the number of cases increased, and large series were studied, opinion began to crystallize that the trauma is incidental to a prior-existing disease, and is instrumental only in an aggravation of the symptoms which then persist indefinitely.

This opinion is probably fairly well established among those who are doing con-

siderable industrial work, as examination of large numbers of spines, as well as other joints, by x-ray, reveals a surprising number of both sexes who present definite changes in bone, cartilage, and fascias, many of whom are entirely without symptoms until they get hurt.

Our own opinion has undergone what amounts to a reversal in the last ten years, and it is with regret that we remember quite a number of cases that we believed were out-and-out malingerers and whom we treated as such, which undoubtedly belong in this class of cases of chronic arthritis, so-called. The classification of these diseases is not at all satisfactory at the present time, as there are probably several different conditions, now grouped under the general head of arthritis, which could and should be differentiated according to the pathological process actually present.

Arial W. George, of Boston, who has had an enormous experience as roentgenologic referee of the Industrial Accident Board of Massachusetts, in a paper read at Kansas City in 1925, proposes a rough classification which seems as good as any:

THE GROUPING OF TYPES OF SPINAL ARTHRITIS

"In approaching the subject of so-called arthritis of the vertebrae, in a broad sense, three general groups appear with more or less characteristic changes.

"(1) The first group, which we do not consider as definitely the result of arthritis, show hypertrophic changes appearing on the margins of the vertebrae, generally in the anterior aspect. These changes are characterized by spur formation without complete bridging between two vertebrae, and may be local to one body or general in all bodies of the vertebrae with possible exception of the upper cervical region.

"(2) The second group are those cases where there are structural changes of the vertebrae or scoliosis with wedging of various bodies and marginal deposits, usually more excessive on the concavity of the curve than on its convexity.

"(3) The third group show changes between the bodies, such as calcification apparently of the intervertebral cartilage and deposits on the margins of the vertebrae. This, as a rule, is general through all the vertebrae excepting the upper cervical region."

In addition, it seems as though the condition described by Fred Albee of New York as myofascitis, should be included.

Etiologically considered, there is a probability that focal infection plays a part in all of these types, for it is the exception to find a workman about mines with a clean mouth and throat. Pyorrhea and apical abscess are so common as to seem normal to these men. In addition, they often suffer from constipation and fecal stasis, in the cecum especially. The diet is almost wholly meat, eggs, bread and potatoes, which will induce arthritis, hypertension and cardiorenal disorders in any human who lives on it long enough. Albee ascribes to these factors the high incidence of myofascial induration, stiffness and pain in injured backs, and has had good results by treating the colonic stasis by irrigations, instillations of cultures of colon bacilli, and the oral administration of alkaline powders. Another etiological factor is undoubtedly the normal reaction of the tissues to stresses and strains constantly repeated, and to postural habits inducing compensatory proliferation; and still another is the chronological factor taking into account those changes induced by age alone. Garvin, writing from the Mayo clinic, states that, in over two thousand radiographs taken mostly for gastro intestinal or genito-urinary diagnosis in persons past fifty years of age, sixty-seven per cent of men and forty per cent of women revealed hypertrophic arthritis of the spine, which may be interpreted to mean that these patients had proliferative bony changes as shown by the x-ray, and that the vast majority were symptomless, and, therefore, that most of the changes were not the result of a true inflammatory arthritis.

The importance of the condition to industry has been emphasized by Louis W. Allard, November, 1929, in a paper based upon his own experience reinforced by a questionnaire addressed to industrial compensation boards, and to thirty-six leading orthopedic surgeons. The consensus of opinion received from compensation boards was that there are a large number incapacitated from back injuries other than fractures, in which the leading symptoms indicate some form of arthritis, but records are confusing and incomplete because medical reports are not made in terms of disease, but of trauma, and the common tendency is to attribute all of the disability to trauma, indicating that a more general agreement among reporting surgeons, as to terms and ideas of underlying pathology, is necessary before statistical information of value may be obtained from this source.

The consensus of opinion from the surgeons seemed to be that arthritis exists very generally among men over forty years of age, and produces disability out of proportion to the injury, ranging from a few weeks to permanent partial disability. From this quite comprehensive study Allard concludes:

"(1) Spinal arthritis is a common affliction of adults, especially the laborer.

"(2) Arthritic subjects are prone to more or less disability from injuries that would not affect a normal person.

"(3) Disabilities complicated by arthritis demand a far longer period of convalescence than similar disabilities in normal spines.

"(4) The victim of well established spinal arthritis is not a normal man. His efficiency is lowered. He is awkward and often distracted by discomfort, easily fatigued, and an easy prey to minor accidents. He is an industrial hazard.

"(5) Patients with recognized arthritis, properly advised, may be retained in selected occupations for years."

Clarence E. Rees, impressed by the long-continued disability and the confusion existing in the industrial commissions' rulings on these cases, suggests active medical treatment with a view to improving the condition of the patients so that they may be returned to some form of gainful work.

Cushway and Maier, of Chicago, reporting on routine x-ray examinations before employment, mention the large number of congenital anomalies of the spine discovered, forty-four per cent, and enumerate 6.3 per cent of cases of proliferative changes at all ages. They conclude:

"(1) Anomalies of the vertebrae are very common and do not necessarily cause painful backs.

"(2) Roentgenologic study of the spine should be made of all employees in industries in which injury of the spine is common.

"(3) This form of examination will eliminate those unfit for certain types of labor.

"(4) Just compensation to the injured will be certain as this form of examination will definitely fix the date of injury within the term of employment."

This agrees well with our own experience, in which we have been so fortunate as to have a large percentage of young men under forty employed, but in late years the men who have grown old in the service are presenting more and more cases of this disease with its consequences, and we believe that we should work steadily to reach a common ground of understanding as to just what this condition is in its multiple forms, or separate entities, as the case may be. We must learn to classify these cases so that selective employment may be based upon sound premises, and cases may be sent up to the industrial commission with some uniformity of opinion as to the part played by arthritis and that played by trauma. In attacking this problem in a practical way, it seems clear to us that every applicant for employment should have a real physical examination, including x-ray of chest, spine and pelvis. That we should try to classify these cases presenting evidence of bony change as infectious, postural and habitual, compensatory, or chronological. The clearly infectious and progressive cases must be excluded. The postural and chronological may be employed in the less hazardous kinds of work, and encouraged to help themselves by dental treatment to guard against infection in the affected joints, dietary changes toward the base, or alkaline, types of diet, and against unusual strains not required in the work they do.

It would be a simple thing to institute so rigid a physical examination as to totally exclude from employment all but selected men, but whether it would be good business is another question, and, so far as I know, it is not the desire of the leaders of industry in the southwest to do so. The industry must bear its full proportion of the consequences of wear and tear inflicted on the men they employ; the great problem is to have it bear equally on industry as a whole instead of on the particular enterprise that happens to be employing a man when his breakdown occurs. And, so far as arthritis of the spine and larger joints is concerned, all the work the man has ever done in his life, every year that goes behind him, has contributed its part to his physical condition at fifty, and it cannot by any stretch of the imagination be construed as an occupational disease, referable to any particular kind of work.

As the contribution of industry, and the general hospitals as well, toward the better understanding of this problem, it appears that accurate records of all cases of spinal arthritis discovered might be kept, with follow-up notes as to the progress and final fate of these patients. An attempt at classification according to the suggestion of Dr. George, and the reporting regularly of the facts ascertained, at our annual meetings, would be little enough for us to do. If the subject were thus kept alive in the minds of all of the men reporting to the Industrial Commission, eventually more uniform opinions might result, which would be of great benefit to the employee, the employer, and to the state.

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INFECTIONS OF THE HAND AND FINGERS

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(Read before the thirty-ninth annual meeting of The Arizona State Medical Association, held at Phoenix, Ariz., April 24 to 26, 1930.)

A human being is the only living creature with a perfectly developed hand; that means man has an organized part of his anatomy capable of many different and delicate movements and so specialized that it can perform the most intricate and difficult work of daily life. Apes and other species of animals can use their upper extremities for some movements like man, for instance, for grasping; but that alone does not constitute a hand by any means. A comparative study of the human hand and the corresponding extremity of the highest developed animal shows great differences of structure and form.

One becomes conscious of the value and importance of a hand only when one is deprived temporarily of its use, for instance, by an injury, and has to carry it in a sling. We are almost helpless without the hand and depend on aid from others for the most necessary and most simple activities. When we get up in the morning with a bandaged hand, we cannot dress ourselves, we cannot wash our faces, we cannot bring the spoon to our mouths and have to be fed; we balance poorly when we walk about and must be doubly careful lest we stumble and fall, without the protection of our outstretched hand. We use the hand constantly from morning till night. All the achievements of the human race are more or less due directly or indirectly to the hands. The words, "handicraft," "manufacture," "handy," "handle," "maintain," "manage," indicate what we achieve in our civilization with that part of our body. In all of these words we find the root word **manus** (hand.)

There are three things which distinguish man from the animal. He is not much different in his anatomical structure and his physiological functions from the beast; but in these three things he differs: **First,** the brain with its reasoning power and free will; **second,** the erect posture and ability to walk; and **third,** the well-developed hand. These discriminate man.

Whatever of art, science and conquest he can boast of, is to a great extent, referable to the hands. He lives by his hands; he sows, he plants, he reaps with his hands. He builds with his hands; he brings forth the resources of nature from the earth with his hands. With his hands he uses instruments to create almost everything that he makes use of in his daily life. The arts, particularly music, are greatly dependent on the highly developed skill of the hand. The violinist, the piano player, the cornettist, all use their fingers in their art. The paintings and sculpture are produced by the hand. It is true that the conception of art is in the brain, but the execution is in the hand.

For the working class, the hand is indispensable. Without the full use of the hand and arm, the working man is handicapped. He earns his daily bread by his brawn. True, the brains are most important and are the chief moving power for progress; but the hands are essential in the work of the world. Considering all these facts, we can understand the importance of the development, and particularly the importance of conservation of this most precious adjunct of our body.

The general practitioner (we all more or less belong in that class) is seldom called upon to treat a condition which can give him more annoyance than an infected hand, nor one whose treatment is fraught with greater difficulties; and perhaps no subject in the whole field of surgery has been more neglected by the surgeon. Even though the results may be the best obtainable, they are never satisfactory from the viewpoint of the patient, who expects the part to be as good as before, and that in much less time than is usually required; while the compensation to the surgeon, for his efforts and skill in preserving, through a tedious course of treatments, a useful though often a somewhat disabled hand or finger, is always more grudgingly allowed than would be that for a total removal of the same part followed by a quick recovery.

The neglect of this subject by the general surgeon probably arises from the fact that few of these cases are referred to him, and, like other people, surgeons prefer to talk or write about that which comes most often under their special care.

A knowledge of the gross anatomy of the hand is necessary to the intelligently successful treatment of any infection in finger or hand.

The most common site for entry of infection is the distal phalanx. Under the nail, filling up the interval between it and the bone and strongly adherent to both, is a fibrous layer—the matrix of the nail. This layer has very slight resistance to infection; once infected, there is no room for the inflammatory exudate to be thrown out. Even very slight inflammatory reaction produces such pressure between bone and nail that the blood supply at that point is cut off or diminished and spread of the infection ensues. This is why infections of the matrix of

the nail are so slow in getting well. The quickest and best way to deal with an infected matrix is to remove the portion of the nail overlying the infected area, and apply a moist, mild antiseptic or saline dressing. Keep the part at rest and change dressings often. Never allow the dressing to become dry before healing begins; thereafter, it is better to use an ointment. If the infection of the matrix of the nail spreads to the soft tissues surrounding the nail, it is usually necessary to remove the nail as before, and with a scalpel make an incision into the infected tissue on each side of the nail. This should be done under anesthesia as it can be properly done **only when the patient feels no pain.** The incision should extend nearly half way from the edge to the fold of the joint. Apply a mild antiseptic dressing, put the finger on a splint and the arm in a sling, and keep immobilized until well.

In infection of the pulp of the finger, the intense pain and throbbing is caused by the fact that joining the skin with periosteum are tough fibrous bands which do not yield readily to swelling. For infection of the pulp the alert physician never waits for pointing of suppuration. An early and free incision, not only through the skin, but down through the pulp, with proper dressings and fixation, will check the spread of the trouble. Incision relieves the tension and it is the tension which causes death of the periosteum, skin and bone, and of all intervening tissues, for the tension soon shuts off the blood supply.

The periosteum over the distal phalanx is often subjected to trauma when the finger is injured. Blood and serum collect under it and these small extravasations are often foci for the development of bacteria lodged there by the blood stream. When bacteria reach such a site the result will most probably be an abscess under the periosteum. The signs are unmistakable. The patient complains of severe pain in the finger, which is constant, throbbing and almost unbearable. The periosteum does not yield readily, and soon a tiny drop of pus has elevated the periosteum from the bone. This process continues until the pus finds an exit, either into the joint or breaking through the periosteum into the pulp. When this happens the relief from great pain is instantaneous and lasts for a few hours, but the pain later becomes quite severe again. Before this rupture of periosteum, there is little or no swelling. Careful examination will always reveal a point of maximum tenderness, which is to be found by pressing gently on the skin with a toothpick or any pointed object. This tender point exists very early in the disease. Under the tenderest point, between the periosteum and the bone, is the seat of the trouble. Over this point is the place to open, and the knife point should reach the bone here only. A large incision is never needed if one can get the patient early. Often only a very small drop of pus is found. As the pus is quite thick, a saline, or Dakin's solution helps to promote drainage. Dress with gauze moistened with these solutions, change often, and keep the part at rest.

When infection following wounds in the skin or subcutaneous tissue spreads, it does so most often by way of lymphatics. The pus can easily spread in any direction. This explains how it happens that pus can extend completely around the finger and not affect the tendon sheath or periosteum at all. It is a point to remember in opening phalegmons of the fingers, for if one cut deeper than the plane where he encounters the pus, and if the incision is over a tendon sheath, he may cut through the fibrous tendon sheath and unnecesarily open its synovial lining. This, too, may follow the insertion of drain or pack, the pressure of same causing necrosis of tendon sheath. The lymphatic vessels pass down the sides of the finger, converge in the web, unite and, crossing the web, enter the palm or pass to the back of the hand. This, as well as the loose nature of the subcutaneous tissue there, explains why it is that the back of the hand undergoes such great swelling in all these infections.

This is simple lymphatic edema—very seldom does pus so spread—but the web between the fingers is the place to look for the first metastatic abscess in finger infections. Bacteria—not pus—get into the lymph channels and, flowing back, reach the web. The fatty structure of the web with its poor supply of blood, makes a good culture medium. Unless incision and drainage by splitting the web right open, is done early, a deep palmar abscess will probably occur. The pain in a web infection is not great and very often is overlooked because of the great amount of pain the patient has in finger, until it is too late to avert a much more serious palmar abscess. We must certainly be on the lookout for web infections in all finger infections, especially those of skin and subcutaneous tissue, if we are to diagnose and treat them early enough to prevent palmar abscess. A little tenderness in the web calls for incision—never wait until the patient calls your attention to pain in this region, nor until fluctuation occurs.

In opening a web, cut straight through it for at least one-half inch and, if pus is not

reached, proceed further toward the palm with blunt dissection. Insert a thin piece of rubber to keep the edges open and dress the fingers wide apart with moist dressings.

The fat in the web comes in actual contact with the tendons of the lumbrical muscles and the common flexors of the finger. Each tendon is surrounded by loose areolar tissue, so it is easy for pus, having once come in contact with a tendon, to dissect its way along it.

Infection of the web of the first space is in a class by itself, inasmuch as it is nearly always intermuscular from the beginning. It follows infection of either the thumb or index finger and can occur by direct extension of pus along perifascial lymph spaces or metastic throuh ordinary lymph channels into the intermuscular space between the first dorsal interosseus muscle and the adductors of the thumb. Tenderness on deep pressure at this point should prompt immediate incision into this space. The incision extends through the skin, superficial and deep fascia until the edges of the muscles are seen. The lines of their fibres cross each other. The space between the two is opened by blunt dissection and the pus will be found there. A drain is inserted to the bottom of the abscess cavity. The whole hand and forearm are immobilized with thumb and index finger slightly separated.

An abscess in the web can readily become a palmar abscess by dissecting its way around the edge of the adduction of the thumb or along the palmar arch or it can spread easily to the wrist along the synovial sheath of the flexor pollicis longus. In dealing with abscess of this web no muscle is cut. Through and through drainage is never used.

We shall briefly consider abscess in the palm. You are told of three compartments. The partitions between the compartments are none of them strong, and pus may pass from any one compartment to any other, for the partitions fail as the bases of the phalanges are approached. The partitions are of fascia and are backward extensions of the deep palmar fascia. The outer compartment lodges the thumb muscles, the inner one the flexor tendons and short muscles of the little finger, while in the middle we have the flexor tendons and lumbricals of the middle fingers, together with vessels and nerves. Tendons and muscles must be surrounded with loose areolair tissue, or they will work stiffly, and infective material spreads readily through loose areolar tissue. The deep palmar fascia is strongest over the middle compartment, and over all compartments the deep palmar fascia thins out, becomes cribriform, and finally fails entirely as we proceed distally. It disappears first in the interdigital spaces, leaving the digital vessels and nerves uncovered by it as they enter the webs of the fingers. Here the superficial fat of palm and web becomes continuous with fat and areolar tissue under the deep palmar fascia. One can thus see how readily a primary superficial abscess can become a deep palmar abscess by direct extension, because the fascia forming the roofs of the palmar compartments is so strong and unyielding, and absesses in any of the compartments may spread well up into the wrist or forearm along the tendons or into its neighboring compartment long before it points on the surface of the palm. The swelling in the palm is, for the same reason, never marked until the abscess is well advanced.

Swelling on the back of the hand is always marked. But this swelling alone should not induce one to incise the dorsum. It is nearly always lymphedema, because, as before mentioned, the lymphatics drain toward the dorsum, but pus does sometimes collect here in the perifascial lymph space, and here, as elsewhere localized deep tenderness is an early and never-failing sign. The tender spot should be opened as soon as found. To "squeeze out" the pus is the most primitive surgery.

How to open a palmar abscess is a real problem and whether to incise vertically or transversely is a moot question. A transverse incision will remain open better while the fingers are extended, and the fingers should always be dressed in extension, but a transverse incision in the lower part of the palm exposes the digital vessels and nerves to danger, while a vertical one higher up imperils the palmar arch. The patient should be anesthetized and an Esmarch bandage applied. One cannot use an Esmarch bandage to advantage unless the patient is anesthetized: hence the need for a general anesthetic and, besides, a local anesthetic in an inflamed area is not often successful. Use whichever incision you prefer, but identify the structures before you cut them. Cut down to the deep fascia and proceed with caution. Divide the deep fascia and then lay down the knife. on deep pressure, go straight on with blunt dissection until the pus appears. Do not pack, but insert soft rubber and fasten it in the wound. Any drainage material that presses on a tendon with any force for even a few hours, may cause local death of the latter. If a tendon, or part of one, dies, it takes four to six weeks to separate and come away. Immobilize the fingers, hand and

wrist, keep fingers in extension and use the hot bath or moist dressing.

Every surgeon remembers the synovial sheaths of the flexor tendons. He knows that there is around each set of flexor tendons, are they lie on the phalanges, a strong fibrous tunnel through which they run and which holds them to the bones. But inside this fibrous tunnel is a synovial bursa, as it were. which surrounds each set of tendons. up to the middle of the palm, while those for the thumb and little finger reach right up to join the bursa under the anterior annular ligament. When pus is found in any of these synovial sheaths, that sheath is to be laid wide open over the place where the pus is, and for at least one to two centimetres above and below this. When the tendon sheath is involved, any attempt to move the tendon causes pain, and there is local tenderness over it.

When pus has invaded the great bursa at the wrist, the anterior annular ligament is cut through and the hand and fingersdressed in hyperextension and left so until healing is well advanced.

When the hot bath is used, the part must be kept on the splint while in the bath, and the solution should be as hot as can be borne without blistering. On no account should the temperature of the bath fall below 110 F. In weak patients it is not well to continue the bath day and night, because of fatiguing the patient; therefore, during sleeping hours the part, still on the splint, is placed in large, hot packs, wrung out of the solution and wrapped in waterproof cover, and the whole surrounded with hot water bags which are frequently renewed.

If an infection of the palm is properly opened and treated as outlined above, the progress of the disease is quickly checked. The drains are removed in from three to ten days and the wound kept open so that pus cannot be retained and burrow. Active motion is encouraged for a few minutes at a time, two or three times daily, as soon as the drains are removed and gradually these periods of activity are increased and lengthened. Only those too long immobilized or improperly handled fail to regain useful function.

THE RELATION OF THE MEDICAL FRATERNITY TO THE STATE INDUSTRIAL COMMISSION

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(Read before the thirty-ninth annual meeting of The Arizona State Medical Association, held at Phoenix, Ariz., April 24 to 26, 1930).

The primary function of the physician and surgeon is the skilled coping with disease and injury. The services of the physician are given upon request and, since most people do not request the services of a doctor until they have become afflicted with some disease or injury, his services almost always embrace the treating and curing of some condition. In this connection, his problem is, first, the proper diagnosis of the condition for the treatment of which he has been called, and, second, the determination of the proper treatment therefor.

The industrial surgeon has the same problem of diagnosing and treatment. In addition, however, he has, in many cases, the additional problem of etiology. In the handling of private cases the etiological side is usually of only incidental character. So long as the physician knows the diagnosis and the treatment, he is not so much concerned about the etiology. When the etiology of a private case does require consideration, it is usually only for diagnostic purposes, because the history of the individual preceding the calling of the doctor often may throw light upon the proper diagnosis.

But in industrial cases the etiology is always important. The etiology of most cases is so apparent and plain that it is not a problem. In the handling of industrial cases the Industrial Commission is vitally interested in correct diagnosis and proper treatment, and equally in the matter of etiology.

A good many of the difficulties that confront the Industrial Commission in the handling of the thousands and thousands of industrial cases that arise each year, are due to faulty diagnosis and ignorance, on the part of the attending doctor, of the etiology of the condition observed.

The impression that one gets is that the average doctor attributes to the Industrial Commission a sort of omniscience in medical matters, and, when a problem is presented, instead of wrestling with the problem himself or doing his best to solve it, the tendency of the doctor is to throw up his hands and pass it on to the Industrial Commission. Then, again, many doctors, if they do give some expression upon a technical point, will make it a very perfunctory opinion, analogous to a "horseback" or "curbstone" opinion of an attorney. Such superficial opinions are usually of but little value and are frequently harmful. If the injured employee is informed of such opinion he sometimes becomes very bitter when his claim is ultimately rejected on the basis of contrary medical opinions.

In such cases the employee never gets the notion out of his head that he is entitled to compensation; and he spends much money

which he would not spend, in consulting lawyers and in legal proceedings which would never have been instituted had it not been for the encouragement and confidence in the propriety of his claim which is impaired by some careless statement or opinion of his doctor. For example, in one case an employee who had sustained a fracture of his leg, developed, some time later, a condition on one of his toes which, when the facts were fully investigated, had no connection whatever with the previous injury which he received. However, one of the doctors, in good faith, wrote a letter to the Commission in behalf of the employee, theorizing as to the possibility of a connection between the injury and the subsequent condition of the toe. It appeared that the real cause of the trouble was a new pair of shoes which the employee had purchased shortly before the second condition developed. Yet the employee had to have a hearing. He appealed to numerous politicians and really caused quite a stir.

However, while it is desirable that doctors refrain from careless expression of opinions, so as not to encourage the filing of claims that lack merit, the most important possibility of improvement lies in the doctors giving more thought, study and cooperation to the matter of etiology, to the end that the Commission may render prompt and correct decisions on medico-legal problems. While the Commission likes to render its work as pleasant as possible, and bespeaks the cooperation of the doctors to this end, the chief desire and aim of the Commission is that its decisions be correct, and also that they be made promptly.

A great number of the cases that arise present medical problems and, strange to say, there are among the medical fraternity many differences of opinion in matters of etiology, which differences of opinion are, in many cases, due to ignorance or carelessness.

If a man goes to a doctor for treatment for a non-industrial condition and later files a claim for compensation with the Commission, the Commission may know that the case is noncompensable. The Commission may have no doubt whatever that there is no connection between the alleged injury and the condition for which the patient is being treated. Yet if his attending physician files with the Commission a report stating that there is a connection, the Commission may not under the law, reject that claim. In such a case, since the evidence is uncontradicted and since such uncontradicted evidence legally establishes the employee's claim for compensation. If the Commission so regards the case, it must, before it can deny such a claim, go to the trouble of obtaining and putting into the record contradictory evidence. In such cases such evidence can always be obtained, because such evidence is correct and proper and just, but it means that the Commission must go to the expense of having the man examined by some other doctor, of a hearing, and of having this other doctor testify. Then, again, it is not pleasant for a doctor to take issue with a colleague in such a matter. And, in addition to all the expense, there is always delay. The reason for this is that the law does not endow the Commission with the power to render decisions, even though such decisions be correct, if they are contrary to the undisputed evidence. Since many claims are presented and are accompanied by medical evidence regarding the etiology, which is incorrect, unsound and illogical, it is necessary for the Commission, before it can reject such claims, to take such steps as may be necessary to place in the record of such case, evidence justifying the rejection of such claim.

For example, if a woman is struck on the breast during her work and a month later is found to be afflicted with a large carcinoma of the breast and the doctor in charge of the case reports that the carcinoma is due to the injury or alleged injury, even though the Commission may have received expert testimony in many other cases to the effect that there could not be an etiological connection between such carcinoma and an injury of such recency, the Commission may not reject such claim without going to the trouble and expense of obtaining contrary expert testimony in the particular case.

If, in such a case, the only testimony in the record, was that of the injured party and her doctor, and the doctor testified that there was a causal connection between the carcinoma and the injury, and if the Commission on such evidence rejected the claim, the Supreme Court, upon review, would reverse the action of the Commission. If, however, the record of the Commission contained the report of another doctor to the effect that there could not possibly be any connection between the injury and the carcinoma, in view of the shortness of the period of time elapsing, then the Supreme Court would uphold an award of the Commission rejecting the claim, because such an award would be supported by evidence.

The point I am making is that there must be, in each individual case, evidence in support of the Commission's decision, whether the decision be in favor of the claimant or against the claimant; and every time the attending physician supplies the Commission

with an opinion upon the etiology which is incorrect, he not only does something which may result in an erroneous decision on the part of the Commission in the event the Commission does not perceive the error in his opinion, but he adds to the delay attending the determination of the case before the Commission and increases the expense of administering the law.

Of course, everybodv makes mistakes. A man who makes very few mistakes but who does very little constructive work is, relatively speaking, more inefficient than a man who makes a great number of mistakes but whose mistakes are less in percentage to the amount of constructive work he does. It is not my purpose to point out mistakes just for the sake of doing so. The purpose of this paper, however, is to point out, if possible, some of the avenues which may lead to increased efficiency on the part of the medical fraternity in its relation to the Industrial Commission.

It seems to me that, while the medical men are cooperating to a great extent in the exchange and the dissemination of knowledge among themselves, such cooperation relates chiefly to diagnosis and treatment, and hardly at all to etiology. Accordingly, while we see the medical fraternity gradually standardizing its practices as regards diagnosis and as regards treatment, we still find much conflict and confusion in the field of etiology.

For example, doctors have very little trouble diagnosing an inguinal hernia, and the treatment of a hernia is usually without difficulty. But, as regards the etiology of an inguinal hernia, we find a chaotic condition. Similarly, as regards orchitis and adenitis. We find that such cases do not cause trouble because of wrong diagnosis, or because of wrong treatment, but we do have much trouble because of what we consider to be erroneous conceptions of etiology. We believe that doctors are not reporting orchitis and adenitis as being due to strains or alleged strains as commonly as they formerly did. This may be due to the fact that the Commission has constantly refused to pay compensation in cases of adenitis or orchitis where the same is attributed to an ordinary strain. Yet we still find doctors reporting such cases, using expressions describing the condition of their patient in language such as this: "Orchitis due to strain while lifting sack of coal;" or, "Adenitis from strain of pushing wheelbarrow." The Commission has refused compensation in such cases because it has chosen to accept as correct the views of those doctors who feel that an ordinary strain cannot cause an adenitis or an orchitis and that employees commonly claim compensation for such conditions when, in fact, they are due entirely to disease.

We have had several cases where hemorrhoids have been reported as due to the strain of lifting and carrying things. We have had other doctors say that they could not be caused by such strains.

As regards hernias, the highest medical authorities have expressed the opinion that a hernia, complete in all its parts, can never arise at the moment of accident or by a single increase of the intra-abdominal tension, be it ever so great; that traumatic hernia is exceedingly rare; that it may occur to any part of the body, but usually not at the site of the normal hernia openings. However, there are many doctors who are testifying in case after case that the hernia was caused by an alleged strain or other relatively mild accident, and a good part of the time of the Commission is taken up trying to disabuse injured employees of the notion that the hernia was caused by a certain strain or accident.

A certain man made a claim for compensation for injury to his foot when a large rock fell on it. While complaining of pain, he returned to his work, nevertheless, upon the doctor's assurance that he had not been seriously injured and no bones broken. He hobbled around, and several months later he claimed again there was something wrong with his foot and an x-ray examination was made at that time, disclosing a large, round callus on one of the metatarsal bones. This was clear evidence that the bone had been fractured. One of the doctors, in good faith, testified that in his opinion the fracture had not occurred from the injury by the rock because an x-ray had been made at that time which was negative. An A.P. and lateral view was made. The x-ray pictures were perfect so far as photography was concerned and there was not the slightest evidence of a fracture showing in the x-ray. The Commission had no doubt whatever but that the fracture did exist nevertheless. It has been proven too many times that such a case is possible. We frequently have cases where a clear x-ray is made and a fracture of two of the metatarsal bones is shown. An x-ray taken subsequently will show callus on three of the bones, from which the doctors infer that there actually had been more fractures than were revealed in the original x-ray.

We receive frequent reports of sacro-iliac slips and x-rays are frequently interpreted as showing sacro iliac slips. Certain ortho-

pedic surgeons of the highest standing have stated that sacro-iliac slips do not occur. One of the doctors said he used to diagnose sacro-iliac slips quite frequently until about ten years ago, but has not made many such diagnoses since and believes that such slips are extremely rare, if they ever do occur. Dr. Albee believes that many of the cases diagnosed as sacro-iliac slips, or sacro-iliac strains, or sacro-aliac relaxation, are really cases where the disability is of toxic origin. He points out that symphysiotomy, pubiotomy, and fracture of the pelvic girdle all strain or separate the sacro-iliac joint to a far greater degree than could possibly result from the trivial injuries that are considered sufficient to produce sacro-iliac strains. Yet, in his experience, the large number of such cases with symptoms referring to the sacro-iliac region had been noticeably lacking.

One of the greatest sources of trouble is the back injury. Unquestionably, thousands of dollars are spent each year for lumbago, arthritis and sciatica. Suppose we have a man whose job is to carry and deliver ice to private dwellings. He has been doing this work for years. Finally he wakes up one morning, telephones his employer he will not show up for duty, and calls the doctor. The doctor calls and finds him running a slight temperature, finds he has a sore throat, infected tonsils and a painful back. The man tells him he strained his back the day previous while carrying ice. In such a case, if the doctor fills out his first report in a perfunctory manner, giving the history of strain of back while carrying ice, and says nothing further, the Commission, relying upon the doctor, will issue compensation checks to that man. It is very likely, however, that that man is suffering from disease and not from injury. Such a case is a typical case. Suppose, instead of straining his back, he claims he strained his wrist, or his ankle, or his knee. If, a few days later, other joints of the body are equally tender and sore, would that not be strong evidence that there was no merit whatever to the claim of injury?

Wherever the disability observed by the doctor does not jibe with the history of incifically call this mattetr to the attention of the Commission. He should do so voluntarjury given him by the patient, he should speily. He should not wait until he is asked by the Commission, because, as stated before, much reliance is placed in the medical fraternity by the Commission and if the doctor does not call the attention of the Commission to the fact that the claim is without merit, the Commission will usually assume that it is a worthy case.

However, we will have employees with infected tonsils and sore throats and running a fever, claiming a strain of their backs or of their sacro-iliac joints, not by reason of any real accident, but by reason of the performance of their usual duties, which may involve lifting and straining, and yet many doctors will classify them as disease. Why should there not be a uniformity of opinion as regards the etiology and diagnosis of such cases?

In another case, an individual came to Phoenix, was examined by a doctor for an alleged injury to his eye consisting of abrading or scratching it with a little twig. He was almost blind in the eye at the time he was examined. I don't recall what the exact diagnosis of the condition was, but it did appear that he was syphilitic. However, the doctor seemed disinclined to ascribe the disability in the eye to the syphilis. A few months later he returned with the other eye similarly affected. It was then decided that the case was never compensable at any time; that the condition in the one eye did not come from the condition of the other eye, but that both came from the syphilis.

In that case the Commission relied upon the doctor's opinion, but I had the feeling that, if the doctor had been a little more familiar with the etiology of the condition than he was, he would have saved considerable expense to the Commission.

In another case we had an individual who was blasted, receiving an injury to his eyes and ears and to one of the nerves controlling the iris. His main complaint, however, was shortness of breath. The attending physician could find no evidence of heart trouble and was unable to explain the shortness of breath but, nevertheless, he attributed it to the injury. The man was 59 years of age. Here again is a case where the doctor was ascribing the disability to an injury with nothing more on which to base his opinion than the claim of the injured man that he did not have the shortness of breath before the injury.

Of course the Industrial Commission cannot expect to find the doctors in full agreement in every case. I recall one case of a negro who was claiming a number of symptoms. There was quite a sharp conflict in testimony of the doctors as to whether it was a brain lesion, or as to whether the case was one of simulation or a neurosis. What I do say, however, is that there seems to be no excuse for such widely divergent views among the medical fraternity as regards medical facts and principles. An ordinary

strain or lifting either can or cannot produce an orchitis. It seems that the doctors should get together and determine once and for all just what the true medical fact is. Then, once that is properly and intelligently determined, the finding should be supplied to all the doctors. If the finding is that an ordinary strain of the employment cannot cause an orchitis, then there should be an end to the constant reporting of such cases by doctors as industrial injuries. The same applies to hernias, to sacro-iliac slips, to alleged lumbar strains, to disability due to conditions on the interior of the eye attributed to dust or other objects abrading or irritating the outer part of the eye, to cancer.

Then, again, the Commission would like to know to what extent the time element is important where an employee claims osteomyelitis of his femur is due to a contusion of his thigh. A case was recently reported where the employee claimed an injury occurred about October 2. The doctor who treated him received a history that he strained his back and right thigh while cranking a tractor, and his treatment consisted of the application of liniment. On November 27 the Commission was advised for the first time that the doctor had determined he had a periosititis which had developed into an osteomyelitis of the right femur. There was at no time any sign of ecchymosis, and the only external evidence of injury was a little red mark which was not observed until two days after the alleged injury and, in the meantime, the employee had applied camphor to the skin, which was considered as a possible cause of the red mark. When one of the doctors saw him, about five or six days after the alleged injury, he found the patient's thigh was two inches larger than the other, but there was no tenderness and no marked rise in temperature. About four days later he had a fever of 102 and a great deal of heat and tenderness over the thigh. On November 11, about twenty days after the alleged injury, a considerable amount of pus was evacuated from the leg. An x-ray was made thirty days after the alleged injury, which revealed extensive osteomyelitis. Shortly thereafter the shaft of the femur broke in two by reason of the inroads of the disease.

Another case which frequently gives the Commission difficulty is the one where an employee develops a cellulitis or an abscess in some part of his body and attributes it to a bump or contusion alleged to have occurred some time previously and in which there is usually no external evidence presented. In such cases we usually find sharp differences of opinion among the doctors regarding the etiology of the cellulitis or abscess.

Back strain, or perhaps I should any back pain, is perhaps one of the principal disabilities which is alleged by workmen's compensation claimants. Usually the symptoms are purely subjective, which adds markedly to the difficulties of the surgeon in deciding whether or not the individual really has suffered a disability. Officials administering compensation laws and the medical directors of such Commissions, are very skeptical of many of the so-called diagnoses of sacro-iliac strains or subluxation, and so-called back strains. If the medical fraternity could develop an increased ability in properly examining and analyzing such cases, tens of thousands of dollars would be saved annually to the employers of the state. The number of cases treated when trauma is not the true cause of the condition, the cost of reviewing medical reports by several different doctors to discover the mistakes made by careless doctors, the employing of medical consultants to help clear up these needless mistakes, and the additional cost of litigation for cases which at first could easily have been settled, all are very burdensome.

Equally as important as the matter of diagnosis, etiology and treatment of industrial cases, is the matter of the doctors' medical bills. We had one case where a man's foot was bruised. There was never an infection nor fracture of any bone. His foot was bandaged up and he was confined to the hospital for ten days; he did not spend his time in bed, but merely was required to be careful not to bump or hurt his foot. For each of those ten days the doctor charged a hospital visit of $3.00.

I doubt whether any state has a fee schedule that is more liberal than the fee schedule adopted by the Industrial Commission of Arizona. I know that the fee schedule of California is from a third to a half lower. With such a liberal fee schedule there is all the more reason why the doctors should cooperate to their utmost to save the Commission and the insurance companies the expense of undeserving claims. The honest physician—and the vast majority of physicians are honest—desires that his patient shall be restored to normal health and usefulness at the earliest moment. If a farmer of moderate means and not covered by insurance recovers satisfactorily, from the doctor's viewpoint, it is certain that the physician will, both for the sake of the man's own welfare, physically and psychologically, and for the sake of his own fee and the reputation for a quick cure, urge that patient to resume his occupation and become a producer again at

the termination of that time. The patient then returns to work, possibly with some slight or moderate discomfort, but he grits his teeth and does his work. The doctor's reputation becomes enhanced by the patient's friends and associates, the doctor gets his fee sooner and more fully, and the doctor has the satisfaction of a job well done, not only physically, but as regards the attitude of the patient. That, I believe, is the normal attitude of the normal physician toward the normal patient.

If the physician has the patient's interest at heart, he will not see that patient, either at his home or at his office, more frequently than is necessary. Nor will he exaggerate the patient's ills or the possibility of future difficulty. What often happens to the same person, with the same income and the same number of dependents, when covered by insurance? Here, altogether too often, the doctor seems to have a different philosophy as to what his conduct, attitude and bill should be. The patient may be hospitalized where, were he not insured, he would not be. Many x-rays are made which, if the patient were standing the expense, would not be taken, and frequently these are made only at the instance of the patient, often in curiosity, since he knows the insurance company will foot the bill. When the point is reached where the employee would have returned to work, the patient demurs. He describes some discomfort that still remains, and then the doctor laxly suggests that some form of electricity might diminish that discomfort. Again the expenses pile up against the insurance company, not only in the bill of the doctor, but particularly in the time lost to the employee.

Why should a patient with a sprained finger or ankle be required to report to the doctor daily when the only result of the call is the fleeting look at the ankle or the wiggle of the finger and the direction that the patient report again the following day? Manifestly, nothing is accomplished for the patient. It is probably very rarely that a physician deliberately prolongs his treatment for the purpose of enlarging his fee. Many physicians, however, with the best of intentions, see their patients oftener than is really justified by the condition being treated. The effect of this is not only a great increase in the medical bill, but it frequently serves to impress the patient with the magnitude of his injury and, instead of hastening his recovery, it tends to retard it.

No one who has had experience in compensation work for any length of time can fail to appreciate the fact that the mental element is just as important as the physical element, as regards the problem of getting the man back to work. A man may be restored physically, as far as medical treatment can assist him, but if his mental impression is that he is still disabled, the effect, so far as the insurance company is concerned, is just about the same as though he were still actually physically disabled. The earlier a man can be put back to work with safety, the quicker will be his ultimate recovery, both from the physical standpoint and the mental standpoint.

PNEUMONOCONIOSIS

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(Read before the thirty-ninth annual meeting of the Arizona State Medical Association, held at Phoenix, Ariz., April 24 to 26, 1930.)

Pneumonoconiosis is the general term for all affections of the lung which result from the inhalation of dust. It includes anthracosis, due to coal dust; silicosis, due to silica dust; chalicosis, to stone dust; siderosis, to metal dust; and byssinosis, to cotton particles and vegetable fiber dust.

There are two important factors in the etiology of pneumonoconiosis. The first is the character and amount of the dust inhaled, and the second is the susceptibility of the individual to its influence.

The two main types of dust are the organic and the inorganic. Since it is very doubtful if the inhalation of organic dust ever leads to definite pneumonoconiosis, the effects of this form will not be considered.

Any inorganic dust is capable of producing definite pathological changes in the lungs if the exposure is sufficiently long. A slight degree of pneumonoconiosis is found in the lungs of all city dwellers who have reached adult life. The frequency and extent of these changes and the length of exposure necessary to produce them, depend on the amount of the dust and its physical characteristics and chemical composition.

In general, dusts appear to be more injurious as their chemical composition differs from the elements of which the human body is composed. Dust from soft material, as coal, does the least damage to the lung tissues. Dusts from shale, slate, iron ore, clay, limestone, plaster of Paris and cement are relatively harmless.

Dust from quartz, quartzite, flint and sandstone is the most harmful. This is due to its content of crystalline silica or silicon dioxide. It was formerly thought that the hard, sharp edges of the silica particles were responsible for the mechanical injury. However, Gardner was unable to produce fibro-

blastic proliferation characteristic of pneumonoconiosis by the use of carborundum, which is almost as hard as diamond and consists of very fine sharp particles. This evidence confirms the belief that silica produces its effect through chemical rather than mechanical action. The finer the dust, the more danerous. Particles greater than ten microns in diameter cannot pass through the finer bronchioles. Microscopic examination of silicotic lungs has shown that seventy per cent of the silica particles have a diameter less than one micron or one-twenty-five-thousandth of an inch. Hence the futility of respirators.

Wherever silica dust is generated and pollutes the atmosphere that workers breath, silicosis will be found, varying with the length of time and the degree of intensity of exposure. Silicosis is seen in workers in granite, sandstone, quartz and gritstone. In gold, tin and lead mining where layers of rock contain a high percentage of silica, silicosis is common. It is also seen in the manufacture of grindstones, in sandblasting, in the manufacture of silica brick.

MORBID ANATOMY

The first effect of the inhalation of dust of any kind, is an irritation producing cough and, later, pharyngitis and bronchitis. After a time the respiratory tract adapts itself to this continuous irritation, the pharynx and larynx become anesthetic and the cough disappears. The small particles which escape the first protective forces of the lung enter the alveoli, where they are seized by the macrophages, and eventually reach the lymph stream. They are then conveyed to the periphery of the lung (the so-called pleural drift.) Due to the insoluble nature of silica particles, they are not dissolved by the lymph but remain as foreign bodies and set up a chronic inflammatory condition. In the end the striking point is the extensive plugging and obliteration of the small and medium sized lymphatics and the compression of the larger ones. This is brought about by the accumulation of dust in these lymphatics and the resulting extensive formation of fibrous tissue. The dominant feature of the pathology of pneumonoconiosis is the fibrosis. In the non-fibrotic portions of the lungs there is more or less emphysema.

SYMPTOMS

It should be emphasized that symptoms do not appear until fibrosis has become advanced. After the mucous membranes of the nose and throat become accustomed to the irritation of the dust, no symptoms may appear for years. Even in the earlier stages of fibrosis there may be very few, if any, symptoms. The rapidity with which advanced fibrosis and its accompanying symptoms appear depends very largely on the amount of silica which the dust contains. Other things being equal, the higher the percentage of silica in the dust, the more rapid and more complete the fibrosis. If there is no silica in the dust, or if it is present in small percentages only, fibrosis takes place very slowly and many years may elapse before definite symptoms arise. In the case of coal dust it takes twenty to thirty years of marked exposure to cause sufficient fibrosis to produce definite symptoms. Potters rarely develop serious symptoms in less than ten to twenty years of constant employment. In pure silica dust, however, where the particles are very small and sharp, and where the dust is concentrated, the average time the occupation can be followed is six and one-half years. Most authorities agree upon an average peroid of exposure of from ten to fifteen years as being necessary for the production of symptoms. American workers place the average period at fifteen years.

According to Jarvis, the first manifestation is loss of appetite. This is followed by cough, apt to be paroxysmal in character, worse in the morning and often accompanied by morning vomiting. Later, sputum usually appears. At first it is small in amount. The color varies from greyish to red, blue, to inky black, according to the character of the inhaled dust. When marked fibrosis occurs, the sputum is often muco-purulent. It may be blood-streaked or small hemorrhages may occur. An associated bronchiectasis may cause abundant sputum. An associated acute bronchitis will greatly increase the amount of sputum.

Dyspnea is the cardinal symptom. It comes on insidiously. Noticeable at first only on exertion, in advanced cases it is apparent when the patient is at rest. In cases of silicosis it is quite an early symptom, usually appearing before the cough and expectoration. Sometimes the dyspnea is asthmatic in character and aggravated by the dust. As the fibrosis advances, the respirations increase and the pulse and blood pressure decrease. The temperature is apt to be slightly subnormal and rarely is above 99.3 degrees. Pleuritic pain is fairly common, as is also a feeling of constriction in the chest. These symptoms seem to be especially noticeable in silicosis.

In spite of all these symptoms the general health may be very little impaired. There is usually a very noticable absence of marked constitutional symptoms. So much is this so that, when marked constitutional symptoms

arise, one should always suspect a complicating pulmonary tuberculosis.

PHYSICAL SIGNS

They are those of extensive fibrotic areas throughout the lung, and of emphysema in the other portions of the lungs. Physical signs of dilated bronchi and bronchiectasis are also often present.

RADIOGRAPHIC SIGNS

The x-ray shadows are quite characteristic. They are those resulting from the deposition of dust and later of fibrous tissue in and around the primary and secondary lobules of Snow Miller. The first stage of silicosis is characterized by an increase in the hilus shadows together with prominence of the trunk and fine branching shadows. During the second state the deposition of dust in the lymph spaces, with the resulting fibrosis, brings about a characteristic diffuse mottling which begins at the hilus and gradually extends throughout the lung, resembling to a slight degree miliary tuberculosis. This so-called "snow-storm" appearance is peculiar to silica dust, for it does not occur when the dust contains no silica. In the advanced stages there occurs a coalescence of the areas of mottling so that it sometimes becomes indistinguishable from pulmonary tuberculosis. However, silicosis differs from tuberculosis in that it is more apt to be bilateral, more marked in the right lung, and usually starts at the hilus and extends to the base.

The relationship of pneumonoconiosis to tuberculosis varies with the different kinds of dust. Very chronic, slowly produced pneumonoconiosis, such as is caused by coal dust, renders the lungs less susceptible to the spread of the tubercle bacilli. There seems to be no doubt that advanced silicosis renders the lung tissue more susceptible to the action of the tubercle bacilli. Health surveys have brought out the fact that the incidence of active tuberculosis among granite workers varies directly with the dustiness of the occupation and the length of exposure. Among the group of hand pneumatic tool cutters with the highest dust exposure (forty to sixty millions of particles per cubic foot of air) there is a very rapid increase of prevalence of tuberculosis with increasing years. That there is a high death rate from tuberculosis among workers in silica dust, has been known for many years. The validity of this observation has been attacked on the ground that a high percentage of the deaths certified as tuberculosis were in reality due to simple silicosis—or a primary and immediate cause of death as silicosis with a contributing cause of tuberculosis—while it is probable that many deaths certified as "fibroid phthisis" were really due to simple silicosis. One has only to study the trend of mortality among granite cutters to realize that not only has the death rate from tuberculosis been rising rapidly but that there is a definite relationship between tuberculosis and the use of the pneumatic hammer for granite cutting. The death rate from tuberculosis among Barre granite cutters has risen from 1.5 per thousand in 1890 to 19.6 per thousand in 1924. Since the introduction of the pneumatic tool, about 1900, with its consequent increase in dustiness, the mortality rate rose rapidly and is still on the increase.

PREVENTION

We have seen that silicosis is an occupational disease due to inhalation of silica dust. If silicosis is to be prevented, steps must be taken to reduce the amount of dust or to remove the dust from the vicinity of the workman, or both. In Arizona copper mines, silicosis does not constitute a health hazard since very little dust is generated, due to the use of the water jet on the pneumatic drills. In many of the large mines in Arizona, the dust hazard is further reduced by the humidification of the air through the regulation of the fresh air intake. This is done by the use of sprays and sprinklers at the fresh air entries to saturate and humidify the incoming air. This method, combined with good ventilation, which rapidly removes any contaminated air, results in greater comfort and increased efficiency of underground workers. In other mines the long tunnels, as well as the ore about to be removed, are wet down.

All men engaged in work where dust is a hazard should be carefully examined by stethoscope and x-ray before being employed. If they present evidence of past or present tuberculosis, or if they present evidence of heart or lung disease, they should be excluded from work. Periodic health examinations, including x-ray examination, should be carried out. Any person showing a definite degree of fibrosis, should be removed from his dusty occupation, for it is well to emphasize at this point that it is only advanced silicosis that is incapacitating and if workmen are removed from the dust hazard before the development of advanced silicosis, the hazard will be greatly reduced.

ARTERIOSCLEROSIS

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(Read before the Maricopa County Medical Society, Phoenix, Arizona, April 28, 1930.)

Arteriosclerosis has of late become a favorite topic of conversation among the laity. In popularity, it rivals sinus trouble and even venerable autointoxication. Everybody seems to know all about it. But when we come to discuss it rationally, we find that we know very little about it. Indeed, some doctors even confuse it with hypertension, despite the fact that the two conditions are entirely independent of each other, though, of course, they might exist in the same patient. Even the name, arteriosclerosis, hardening of the arteries, is misleading, since it gives no indication of other very important changes preceding the sclerosis. Thanks to Adamy, Monckenberg and, especially, Aschoff, we are able to get some idea as to what those changes are.

What, then, is arteriosclerosis? Before attempting to answer this difficult question, let us briefly review the structure of the normal artery. It is customary to divide the arterial wall into intima, media and adventitia. The intima, the inner layer of the artery, consists of a layer of flat endothelial cells which lines the entire vascular system, including the heart, a fibroelastic layer and an internal elestic membrane. In the aorta, the intima is thicker, containing in addition an inner elastic layer, an outer muscular layer, the two separated by elastic stia terminalis, The media is made up of flat bundles of involuntary muscle, separated by elastic fibers, and of an external elastic membrane. The proportion of muscle and elastic tissue varies greatly in the different arteries. In the aorta and the other large arteries the elastic fibers predominate. This gives them their great elasticity, enabling them to perform their function as distributing channels and maintaining a steady flow of the blood stream, even during diastole. In the arteries of the internal organs and extremities, the muscular tissue predominates, allowing for greater distention and contraction, in accordance with the demands placed on them by the given organ. The adventitia consists of fibroelastic tissue which blends with the areolar tissue surrounding the artery. The nutritive blood vessels are located in this layer. They supply food to the adventitia and the outer half of the media. The inner half of the media, as well as the intima, is nourished by the blood stream within the artery.

Aschoff recognizes three stages in the life of an artery: ascending, the summit and the descending. During the ascending stage, all the constituents of the wall of the artery increase equally. During the summit, which begins at the thirty-third year, the artery remains stationary. During the descent, beginning at the forty-fourth year, the elastic tissue decreases and the connective tissue increases constantly. This is one of similar changes taking place during this period in any other part of the body possessing elastic tissue. It is most noticeable in the skin, where the smoothness maintained by the elastic tissue gives way to the wrinkles produced by connective tissue. In the arteries, this change diminishes the elasticity and increases the diameters of the vessels. The increase in the transverse diameter causes dilation of the blood vessel. The increase in length accounts for the tortuosity of the artery. Acshoff describes a simple experiment to show what age does to the elasticity of the aorta: He detaches the aorta from the posterior wall between its two ends, and cuts it in the middle. The cut ends immediately retract several centimeters. In the aged, no retraction takes place. These changes occur in all and are physiologic. They constitute fibrosis of the arteries, but not arteriosclerosis.

Then there are the atheromatous changes which are not physiologic. They occur in all ages, though more frequently in infancy, puberty and old age. They are seen mainly in the aorta and other large vessels where great strain is used by the vessel, as near the origins of the vertebral arteries and the carotids and iliacs. At those points, white patches are seen which vary in size and consistency. In infancy and puberity, the process begins with a deposit of fat in the cement substance of the fibroelastic layer of the intima. This deposit separates the elastic layer and pushes up the endothelial lining of the intima. As a reaction to this invasion, a few wandering cells appear and some proliferation of connective tissue takes place. The process does not ordinarily go any further. In time, the connective tissue and the deposit are absorbed and the intima is restored to health. Not so in the aged. Though here, also, the atheromatosis begins in the intima by a deposit of fat, the process goes on unchecked to more serious changes. In the first place, the character of the intima is different from that in the young. The elasticy is gone, connective tissue is in abundance, the dilated artery offering an easier access to the onrush of the invading fat. The reaction to this invasion is correspondingly more marked, with the result of still

more increase of round cell infiltration and connective tissue proliferation. This process is no longer limited to the inner half of the intima. It spreads both ways and invades even the media. And when the place becomes too crowded, hyaline degeneration and necrosis of the connective tissue cells take place. This further complicates the situation. Precipitins put in an appearance. They are meant to get rid of the mess. Instead, they make matters still worse. They break up the fat, liberating cholesterol. This is acted upon by the fatty acids, producing soap, mainly of calcium, resulting in hardening of the arteries. The proper name for this process is not arteriosclerosis, but atherosclerosis. And the two important elements in its production are atheromatous changes superimposed upon previously changed arteries of old age.

As to the nature of atheromatosis, Virchow attributes it mainly to a mechanical cause. He first establishes the fact that fat found in the patches is not a degeneration but an infiltration. He also noticed that these patches are usually found in spots where the arteries show greater strain. He therefore concluded that the process is caused by the plasma pushing its way into the intima in a little greater volume than is necessary for the vessel's nourishment.

Aschoff's investigations led him to believe that, in addition to strain and the inbibition theory, as he labeled it, there must exist some dyscrasia in the blood which causes some physiochemical changes in the fat metabolism. First, it was noticed that, during the World war, German soldiers began to show continuously less atheromatosis as the war progressed. That would be explained by the fact that their fat rations grew less. Then it was shown that rabbits fed on cholesterol developed atheromatosis, and that, by withdrawing cholesterol, the atheroma disappeared. Similar results were obtained by feeding rabbits on lanolin. When thyroid gland was added, no atheromatosis took place. This explains why atheromatosis is more common in infancy, puberty and senility, as in each of these stages there is an unbalance of fat metabolism.

To sum up:

When arteries are working under strain, and there is a certain unbalance in the fat metabolism, atheromatosis will take place. If this process occurs in senile arteries, the changes become more serious and end in otherosclerosis.

The various affections of senility are due to the impaired function of the arteries. Arteries are built to carry food and oxygen to every cell of the body. When the architecture of the artery is changed by fibrosis, the function as distributing agent is impaired. The cells, not receiving their full ration, will not be able to perform their respective duties properly. As the changes in the arteries continue to obstruct the flow of blood, the cells continue to starve. Less useful work is performed by them and more waste products accumulated in the body. These have a deleterious effect on the already degenerated arteries. A vicious circle is formed.

Symptoms produced by atheromatosis are an intensification of the above. Life becomes a burden, and at last reaches a state where it can no longer continue.

Not all the arteries are necessarily affected at the same time and with equal intensity. This accounts for the difference in the ability of the various organs to carry on. The organ that is supplied by a softer artery will naturally be in a better position to continue its life work.

The etiology is not well understood. Some text books still talk of alcohol, tobacco, coffee and other "vices" as causes of arteriosclerosis. But these are mere guesses, as none of the above-mentioned causes was proven to be able to produce hardening of the arteries. On the contrary, we know of many who indulge in them and retain their arteries, retain their normal elasticity, while others who led an exemplary life have arteries as hard as a rock. Aschoff teaches us that a certain blood dyscrasia is necessary to produce atherosclerosis. But he does not claim to know what factor brings about that dyscrasia. A good guess is that there is no cause; that this is nature's way of getting rid of us. The reason these changes differ so markedly in various individuals might be explained by hereditary disposition. But this also, is only a guess. At times, we might have to hide our ignorance in the presence of some patient for his own good. We then might be permitted to let our imagination work. But, among ourselves, let us be honest and admit that we do not know.

As for treatment, since we do not know what causes the condition, we cannot be expected to know what to do for it, either prophylactic or curative. Here, again, if we deal with a neurotic old maid of either sex, we may have to pretend that we know all about it. Otherwise, aside from symptomatic treatment, we do not know what to do about it.

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ABNORMAL EYES IN CHILDREN OF GRADE SCHOOL AGE

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The large number of cases showing abnormal eyes, seen at the Clinic of the Social Service Center, suggested a survey among grade school children to determine the number of abnormalities. Conditions considered as abnormalities include not only errors of refraction, but also cases of infectious eye diseases, such as trachoma and acute epidemic conjunctivitis. The actual number of infections must be an approximate one, as it would vary with diffreent surveys taken at different intervals of time. However, in examining the past records it is found that this number is fairly constant throughout any one year, and the variations are most marked in comparing different years.

The method of examining for visual defects in children, in Phoenix, is as follows: At the beginning of the school year the school nurse examines the children for any deviation from normal. Records are kept on all cases examined and children with sufficient defect are advised as to medical attention. A second routine examination is attempted shortly before the end of the school year. In the interval the school nurse spends a certain amount of time each week at each school, depending on its size and the number of schools she has under her care. During these visits she rechecks those defects previously noted, gives treatments and sees any new cases brought to her attention by the teacher.

At the present time every state has some form of legislation governing school nursing and almost all of them include an examination of the eyes as routine. The first organized effort to advance this supervision was "The Association for the Conservation of Vision," which was formed as a Division of the Bureau of Public Health and Instruction under the American Medical Association. This was formed in 1913 and the results of its efforts were immediately gratifying. Previous to 1913, only eight states had laws relative to the examination of grade school children, beginning with Connecticut in 1897. Within the two years following 1913, ten more states were added and since that time every other state has passed some form of this legislation. Several other organizations have helped to promote this legislation, notably The Association for the Prevention of Blindness."

Six Phoenix schools were checked for this survey. They were chosen as representative of various types of pupils and home environment and included two schools having white pupils, one with mixed white and Mexican and three Mexican. The largest school was Emerson, with 1,100, and the smallest Douglas, with 130.

Emerson is a school admitting only white children and, of its 1100 pupils, 600 were examined this year; slightly more than fifty per cent (54.5.) Of this number examined, 497 had normal vision—considered in this case to be 10/10—leaving 103 with some degree of visual defect. However, this percentage cannot be applied with accuracy to the remaining 500 children not examined, as the initial examination included all the children in the entire school with obvious visual defects, such as blindness in one eye or such a degree of impairment that there was difficulty in doing the usual tasks at school. With this provision we shall consider only the 600 examined.

Of the 103 defects, sixty-four had normal vision in one eye with vision in the other eye not less than 8/10. This group was considered to have adequate vision and were not advised to have corrective measures taken. Sixteen other children had vision equivalent to 8/10 in each eye and this group was also considered to have sufficient vision. It was found, therefore, that thirty-three children had visual deficiency sufficient to advise correction. In this group one child had 10/10 vision in one eye and 6/10 in the other eye; another 10/10 and 2/10; another 6/10 in each eye and the others had less vision down to entire blindness in one eye. Two children had strabismus of extreme degree. Twenty of the thirty-three children have already had corrective measures taken, and this number includes all those with the more marked defects.

Emerson school shows a low number of trachoma cases, six receiving treatment at the present tmie and two having had their lids rolled. The number of other cases of eye disease fluctuates so much that a compilation at any one time is valueless. The school nurse states that an average number would be less than a dozen.

The other school with only while children was McKinley, where the number is 250. Here 160 were examined or sixty-four per cent—of whom 124 were found normal. Of the remaining twenty-six, only ten had visual defects sufficient for correction, eight of these having minimal defects, one having 2/10 in one eye and 10/10 in the other, and one having one blind eye and 10/10 in the other. Eight of these ten have been corrected, which is a good record.

At McKinley, five cases of trachoma are being treated and four cases have had their lids rolled. In addition, there is at present one acute trachoma barred from school and under treatment. This school is almost free from acute infectious conjunctivitis or other eye disease at this time.

Grant school has the largest enrolment of Mexican children, with 650. Sixty, or about ten per cent were examined in the fall or first examination, and, of this number, nineteen had normal vision or 10/10, and an additional twenty had 8/10, or sufficiently normal vision not to require correction. The twenty-one children with visual defects sufficient to require correction ranged from fifteen with the equivalent of 6/10 vision, to children with maximum vision of 3/10. Only two of this group were corrected. Due to the incomplete report, this school cannot be compared to the preceding schools reported. However, it is seen that the percentage of correction of defects is much lower at this school.

Trachoma is much more prevalent at Grant school than in the preceding two schools reported. There are forty-five cases here, and, in addition, the number of cases of infectious conjunctivitis is usually over fifteen.

At Lincoln school, fifty children, or twenty-five percent of the 200, were examined. This school has Mexican children. Forty-two of the fifty examined had normal vision, and, of the remaining eight, only one had vision deficient enough to require correction.

There are two cases of trachoma at Lincoln school who have had their lids rolled and one other is under treatment at this time. This school has a good record also in the low number of cases of acute infectious conjunctivitis.

Lowell school has both white and Mexican pupils, with an enrolment of 800. Two hundred ninety, or thirty-six per cent, were examined and twenty children were found to have vision low enough to require correction. These defects vary from five with 6/10 vision in both eyes to three having one blind eye and the other normal. Fifteen, or seventy-five per cent of those with considerable defects have been corrected.

There are at present fifteen cases of trachoma under treatment, with four cases operated. This number is high as compared to the figures for Emerson and McKinley. Lowell also has a higher average number of acute infectious eye diseases.

The treatment of infectious eye diseases falls largely upon the school nurse and the teachers, as a majority of the Mexican children will receive no home treatment. In this work, many of the teachers are very efficient and are of great help. It is unfortunate that most school nurses have too many children to care for so that daily treatment falls upon the untrained teacher. An example of this hazard was recently seen when a teacher in one of the outlying schools received a severe acute conjunctivitis while attempting to treat one of her pupils with a similar affection.

DIAGNOSTIC FORUM

CASE OF CHRONIC PAINLESS JAUNDICE

(Case 15181, Case Records of Massachusetts General Hospital, from New Eng. Jour. of Med., May 2. 1929, p. 939.)

CASE HISTORY

An Irish-American, forty-six years old, an inspector in the fire department, entered January 8 complaining of jaundice.

A year before admission he began to have colicky pain in the lower abdomen. worse at night, intermittent and quite severe, relieved somewhat by plasters. He had anorexia and mild nausea. Two weeks later, an hour and a half after going to work, he felt nauseated and vomited some greenish bile stained material. He was in a state of collapse. He went to a hospital, where he was told that a blood test raised a suspicion of syphilis. A second test was negative. His skin was yellow. The following day he went to a specialist. Gastro-intestinal x-rays were made and he was put on a diet of milk and cream with powders for six weeks. During that time his weight fell from 169 to 149 pounds. During the period of treatment he had no symptoms except jaundice and itching of the skin. The following March he had an attack of sharp colicky pain after every meal. He went to another hospital, where operation was done March 28. After it he was told that he would live only six weeks. He was privately informed however that even after the operation they were not sure of the diagnosis. Kahn and Wassermann tests were both positive. A Graham test showed no filling of the gall-bladder. He was discharged April 14 weighing 143 pounds. He was told he might eat anything and should return for x-ray therapy. On April 17 he came to the x-ray clinic, but was advised to wait for two weeks because treatment at that time might stir up complications. He did not return. He continued to work, although not so hard as previously. His appetite was good and he ate everything. His stools were clay colored at times. His urine was very dark. The jaundice persisted. The itching of the skin stopped. Three weeks before admission he had an attack of indigestion, with gas and distention. A physician found fatty stools and told him some of his food was going through undigested. Since that time he had eaten less and had felt relieved. At this time he noticed that his feet were slightly puffy. For two weeks before admission he had had a mild cold with unproductive cough. At admission he had persisting jaundice, slight loss of strength, steady weight, some mental depression and lack of energy. His abdomen was somewhat distended. There was a questionable hernia in the wound. His urine was dark colored. He occasionally urinated once at night.

His past history was negative except for left inguinal hernia repaired twenty-five years before

admission at this hospital, and gonorrhea and soft chancre twenty-three years before admission treated by a syphilologist of this hospital. Until sixteen years before admission he took a moderate amount of alcohol. He smoked a package and a half of cigarettes and two or three cigars a day.

His mother died of shock.

Clinical examination showed a very much wasted man. Skin dry, loose, scaly; moderate icterus. Teeth carious. Apex impulse of the heart not seen or felt. Sounds very faint, distant and of poor quality. Rhythm regular. Pulses and arteries normal. Blood pressure 90/40. Right upper chest dull, with bronchial breathing. Breath sounds loud and exaggerated throughout. No rales heard. The abdomen showed the scars of the previous operations, with a bulge of postoperative hernia in the right rectus scar. A hard irregular mass the size of a grapefruit in the epigastrium. A nodule the size of a marble in the skin above the umbilicus. Below the right costal margin two finger breadths from this mass was a smooth, dull, movable mass the size of an orange extending down to the right lower quadrant. Rectal examination showed hemorrhoids, no masses. There was marked pitting edema of the ankles and feet.

Before operation urine normal; blood not recorded: icteric index 50 to 75; van den Bergh test 16.12 milligrams of bilirubin per 100 cubic centimeters of blood: clotting time 14 to 19 minutes.

Before operation temperature 98° to 101.3°, pulse 80 to 110, respirations normal.

January 14 operation was done. Two days later patient was in poor condition, had difficult and increasing respiration and was running an irregular low grade temperature. Both bases were filled with coarse bubbling rales. He was very deeply jaundiced. He continued in the same condition and on January 18 died.

DISCUSSIONS

Diagnostic conclusions offered on this case by members of the Phoenix Clinical Club were as follows:

Dr. A. C. Kingsley:—Syphilis of liver; tumor of pancreas.

Dr. Howell Randolph:—Carcinoma of head of pancreas, probably secondary to carcinoma developing in duodenal ulcer; course too slow for primary carcinoma of pancreas; carcinoma of bile ducts unlikely; ulcer of duodenum with rupture and adhesions a possibility; metastases to liver and lung.

Dr. J. M. Greer:—Carcinoma of head of pancreas.

Dr. Victor Randolph:—A diagnosis in this case can be only a guess; carcinoma probable, involving hepatic duct by pressure and mesentery and with subcutaneous metastases; primary may be in pancreas, stomach or cecum; infrequency of abdominal metastases from primary liver or bile duct carcinoma rules these primary sites out; syphilis also present.

Dr. T. T. Clohessy: Gastric carcinoma with metastasis in liver.

Dr. Fred Holmes:—Carcinoma of bile ducts or gall bladder, or head of pancreas; metastatic carcinoma in lung.

Dr. H. P. Mills:—Cancer of pylorus with metastasis.

Dr. F. C. Jordan:—Cancer of gall bladder or bile ducts.

Dr. S. I. Bloomhardt:—Carcinoma of pancreas; secondary of pylorus and possibly of right lung; terminal pneumoniai; syphilis.

Dr. O. H. Brown:—Important record points; epigastric tumor, mass to right, jaundice, negative Graham test, downward course, operation, doubtful prognosis, Wassermann positive, indigestion, pruritus, carious teeth, denied x-ray treatments, feeble heart, swollen feet, lung changes, fever.

Diagnosis: Cancer head of pancreas; enlarged gall bladder; myocarditis; syphilis; bronchopneumonia; bilirubinemia; carious teeth; indigestion; pruritus; biliary obstruction.

DR. W. WARNER WATKINS

The presenting symptom in this patient is jaundice. Jaundice is an exceedingly interesting study, and I wish I had time to go into its biochemistry and the clinical significance of its various aspects. Jaundice is the name given to the appearance of bilirubin in the visible tissues of the body,—skin and mucous membranes, etc., on account of the failure of its eliminatioin through the liver.

It has now been conclusively proven that bilirubin is not formed in the liver, but is simply eliminated by that organ, in a manner comparable to the elimination of urea by the kidneys. The suggestion that bilirubin is not formed by the liver was suggested as long ago as 1847 by Virchow, but the final proof of this has been slow in coming. Bilirubin is derived from hematin,—the iron bearing portion of hemoglobin; bilirubin is chemically identical with hematoidin which is the blood derivative which stains the skin yellow after a bruise. Bilirubin is formed by the reticulo-endothelial cells, chiefly of the bone marrow and the spleen, and is eliminated through the liver cells. In passing through the glandular cells of the liver, some alteration is effected, so that the bilirubin appearing in the bile ducts differs in some not entirely known fashion from that which circulates in the blood. The difference can be detected by laboratory test (Van den Bergh) allowing us to say whether the bilirubin present in the blood plasma has passed through the liver cells or not.

Jaundice due to obstruction to the normal passage of the bile through the bile ducts occurs because of the reabsorption of the bilirubin into the blood, and this gives the direct Van den Bergh,—which indicates obstruction in bile tract somewhere. Jaundice due to retention in the blood of bilirubin formed by hemolysis of red cells, by red cell destruction, by sepsis, infectious jaundice, Weil's disease, or by anemias,—that is bilirubin which has not yet passed through the liver cells,—gives the indirect Van den Bergh reaction which indicates hemolytic type of jaundice. The diphasic reaction is where both reactions occur, and indicates that both types of jaundice are present.

The three common causes of obstructive jaundice are gallstones, cholecystitis without stones, and cancer in or about the bile tract; less common causes are cirrhosis and syphilis of the liver. The common causes of hemolytic jaundice are acute yellow atrophy, pernicious anemia, sepsis and other infections, and familial jaundice.

Our patient has a past history which indicates that he had syphilis. A year ago he had pain, with anorexia and nausea, followed by jaundice, and then loss of weight. His Wassermann was questionable, and was probably one of those plus-minus aggravations which we get in jaundice. What are the indications from the history? The hemolytic types seem to be ruled out by the symptoms, which point plainly to one of the obstructive types. The Van den Bergh is not mentioned but should have given a direct reaction. We have as possible causes, cholecystitis with stones, cancer in or about the bile tract and syphilis of the liver.

Gastro-intestinal x-ray evidently showed some sort of stomach defect which caused them to put the patient on ulcer regime for six weeks, during

which time he lost 20 lbs. Then they operated upon him and found some lesion which they regarded as cancer, but were not entirely positive. They probably did some sort of palliative surgery on the stom ch, as he had some relief but jaundice continued. Wassermann and Kahn tests are positive. so stated. Graham test showed no filling of gall-bladder. As you know, this test aims to fill the gall-bladder with bile impregnated with a dye more or less opaque to x-ray. When the gall-bladder is able to empty and refill during the period of elimination of the dye, we get a shadow of the organ on the x-ray film. If this process of emptying and refilling is interfered with from any cause, we will get what was found in this patient,—negative findings.

Their prognosis was evidently hopeless, because they took off dietary restrictions and suggested x-ray therapy.

At the present examination, it has been nine months since his operation. Jaundice has persisted, weight has remained steady, though there is loss of strength and failing memory,—which jaundice will cause. There is a mass under the umbilicus, which is probably tumor, and another one under costal margin which is probably distended gall-bladder. Is there anything else which comes into the picture than cancer? I cannot think of anything reasonable. The remaining question of interest is the primary origin of the cancer. Cancer in this region, producing jaundice might arise from liver, gall-bladder or ducts, pancreas, stomach or duodenum. Whatever he has caused some filling defect in stomach nine months ago; this with the character of symptoms seems to rule out primary liver cancer. Course of the disease would seem to me to be against primary cancer of the pylorus. The balance of probability is in favor of cancer arising from head of pancreas or from the bile tract, and it is purely speculative which.

The last surgeons evidently would not take the diagnosis of the first ones,—since they were so far off in their prognosis of six weeks life. So they went in to see for themselves; I judge they went in and backed out.

My diagnosis is cancer of the head of pancreas or of the bile tract primarily, with secondary involvement of stomach and liver, possibly lung. Terminal pneumonia.

DR. R. J. STROUD

CAUSES OF JAUNDICE

1. Obstructive: Cholecystitis, cholangitis, carcinoma of ducts, pancreas, stomach; adhesions of ducts from duodenal ulcer, stone of hepatic or common ducts, massive stone of gall bladder.

2. Non-obstructive: Weil's disease, catarrhal jaundice, secondary to acute infections as typhoid, etc., syphilis, acute yellow atrophy, cirrhosis, acute and chronic hepatitis, carcinoma of liver, antisyphilitic administrations, tuberculosis, hemolytic jaundice.

This classification is really not tenable but gives a working basis for diagnosis.

Most of the non-obstructive forms are painless, but carcinoma of the ducts and large stone of gall-bladder may also be painless.

Exclusion is made, because of subsequent history, of all but syphilis, carcinoma of some organ, cirrhosis, and perhaps cholecystitis with large painless stone.

There is no history which would lead to the diagnosis of cirrhosis except the aclohol, but there has not been described either a gradually growing mass with extension of liver dullness evenly, and the later masses hardly give the picture. There is no ascites. He has a positive Wassermann and Kahn. Syphilis generally causes jaundice in the primary cases, and gumma in the tertiary stage (I saw a case cleared up under arsenicals, although the skin showed irritability to arsenic); syphilis as a cause is never entirely ruled out. He was treated for a stomach ulcer or duodenal ulcer following x-rays. However, the treatment with alkalies may have been for gall-bladder disease. His later symptoms of pain after meals point more to gall-bladder trouble or irritation or dysfunction for some reason rather than the ulcer.

Cause for operation is not known. It looked as if he was opened up and closed with suggestion of carcinoma, either of the stomach, pancreas, gall-bladder or liver.

He was advised to have x-ray therapy for the condition. I do not know what for. General malignancy of the region is too close to other organs for the roentgenologist to care to give exposure. Maybe they thought they would activate a syphilis. His gall-bladder did not show filling; something was in the way; a growth or obstruction or chronic inflammatory closure of the ducts. At first, the appetite was very good. Later and at the same time he found that by eating less, he felt relieved; a good sign of carcinoma of the stomach, where the stomach generally takes small amounts of food, and soft food. I would like to know if soft foods were better taken.

Constipation was not mentioned. All the signs of dark urine, fatty stools, clay-colored stools, high icteric index, clotting time, and Van den Bergh, mean that the bile is being excreted through the kidneys, and the blood is carrying a big percentage of bile elements, and that the intestines lack bile.

On entering the hospital, he showed signs of a debilitating disease which caused the low blood pressure and signs of a failing heart.

The large grape fruit sized mass in the epigastrium was a new growth; the smaller smooth mass could have been a gall bladder ,either with a stone, or malignant, or it could have been some growth of the intestines without giving any bowel signs. More suggestive of carcinoma of the gall bladder.

After operation he did not rally for his physical condition could not sustain the shock. Heart then failed rapidly with wet bases of the lungs. Jaundice increased rapidly (we do not know what was done; maybe an exploratory only). An extensive operation with such a blood pressure was dangerous and perhaps not attempted. Normal urine before operation did not mean that there was no bile. There must have been bile with those blood findings.

He was jaundiced before Wassermanns were done, and yet he was treated for so-called soft chancre when younger. No history relative to antisyphilitic jaundice is given. May have taken mercury at times.

His original operation puzzled the operators. He was first told he would die in six weeks; then that they were not positive in diagnosis. It calls for care when the patient was not even seen.

The early history of carcinoma could have been compatible with the pain in the lower abdomen, then followed painless jaundice, with later a mass,—two masses ,and a possible extension to the upper right lung which could be metastatic.

Where was the carcinoma? We have stomach history and treatment after x-ray. Pancreas could give the picture but with such a mass one would expect sugar signs, although the head could bear the whole brunt of the burden.

Diagnosis: Carcinoma of gall bladder with later extension to liver, stomach and lungs. 2. Carcinoma of stomach, primary. 3. Syphilis. 4. Cirrhosis.

Discussions of this case by Group 1, Yavapai County Medical Society and Medical Officers of Fort Whipple, at their regular meeting on Feb. 25, 1930, were as follows:

DR. C. R. K. SWETMAN, Prescott, Ariz.

We have a case of a man forty-six years old who begins with gastro-intestinal symptoms, particularly jaundice. I want to call your attention to the fact that jaundice is really the presenting symptom.

To begin with, he had some colicky pains, loss of appetite and mild nausea. He vomited once only, at least we are told of only one time. He went to the hospital and the following day had a gastro-intestinal series done and was put on a Sippy diet. For six weeks he was on this diet, felt fairly well but continued to lose weight,—lost twenty pounds in that six weeks. The fact that he felt well was probably due to the fact that he was on so light a diet.

Within three months or less of his first symptoms he had enough pain and other symptoms that he went to another hospital and was operated. We are not told, of course, what that operation was, but evidently it gave him some relief; it relieved the itching from the jaundice. Again I want to stress that the jaundice, which was severe, was one of the first and most persistent symptoms. I found one interesting point,—that jaundice accompanied by severe itching is due to obstruction of the ducts, not to anything in the liver itself. If this man of forty-six has obstruction of the duct we want to know first what it is. We have, in the little period following operation, some hint as to what it is. He had clay-colored and fatty stools, which point to the pancreas. We are told that his urine was normal but, at the same time, another man observing says the urine is very dark colored. We take that to mean it was normal as far as showing any inflammatory condition of the kidneys is concerned, but we do not know whether or not it contained bile. With icterus and so much bilirubin in the blood, we think that dark color means bile in the urine, which is good evidence of obstructive jaundice.

There is another interesting point; after the first operatioin he was told he would not live more than six weeks. Another man told him that even after the operation they were not sure of the diagnosis. We find that that is rather typical of conditions in the head of the pancreas, as even after opening the abdomen it is very difficult to tell whether the enlargement in the head of the pancreas is inflammatory or malignant.

Another thing that is interesting to us is the early jaundice. If it is a conditioin in the head of the pancreas, how account for it? In two ways. If it is malignancy it would be very soon likely to cause some secondary or metastatic condition around the gall ducts themselves, but the common bile duct lies in a groove in the head of the pancreas and, in about two-thirds of cases, is entirely surrounded by pancreatic tissues for variable distances. The sounds in the heart, we are told, were weak and distant, the pulse regular but also weak, and blood pressure only 90/40. We believe that this indicates a failing heart muscle, simple weakness. We are not told that there is any murmur or abnormal sound, in fact the sounds were so weak they would probably not be picked up if there. He had pitting edema of the ankles. Pitting edema means fluid, of course, in the tissues, and that was probably due to pressure from this malignant growth which we believe he had.

He was operated and the operation did not do him any good. We are not sure what that operation was but it may have been only exploratory or it may have been drainage of the gall bladder. However, he continued to fail very rapidly and lived only four days after the operation was performed.

In the history we are told that a positive Wassermann and Kahn was discovered at another hospital. However, it strikes us as very significant that when he came to the Massachusetts General they did not think enough of this to do another Wassermann on him. They just let it go.

In yellow conditions of the skin we always, of course, think of pernicious anemia, but there is nothing in this case to even suggest that.

DR. J. T. MALONE, Whipple, Ariz.

Continuing our differential diagnosis, we are confronted with several possibilities. A man of forty-six with sudden gastric symptoms would most certainly lead us to consider malignancy, but the history tells us definitely that this man vomited only once. The pain is not the picture of pain in gastric malignancy; the symptoms were too variable.

Our first impression, when he was suddenly taken with this pain, was that it was an acute condition of some kind. Acute pancreatitis, most authorities tell us, is something that can not be diagnosed unless operation for acute abdomen is performed.

We have a man who gives us a picture of shock. He is suddenly seized, is nauseated and vomited. We think that the interne was rather misled in taking the history and that it was general weakness and not surgical shock, because we find him going the next day to a gastric specialist and having x-rays taken. They were certainly suspicious of some involvement around the pylorus, some suggestion of peptic ulcer, and on that basis he was put on a Sippy diet. He lost twenty pounds. If this condition was non-malignant, the peptic ulcer would have improved, symptoms become less and he would not have lost so much weight.

It is generally known that jaundice, especially of the catarrhal type, is always accompanied by sudden loss of weight, from twenty to thirty pounds.

A little later we have a recurrence of these symptoms; from January to March. At this time the surgeons probably thought an exploratory operation was indicated. We can not definitely predict what was found at that operation, but on the basis of one sentence in the history, one of three things was done; gall bladder was drained, which would account for lessening of the itching; cholecystenterostomy; or gastro-enterostomy, presuming that there was ulcer at that time. We are inclined to believe that the gall bladder was drained into the gut.

As we go along a little farther and consider conditions outside the gastro-intestinal tract, we think of hypernephroma. We are told the diagnosis of hypernephroma is based mostly on urinary findings. The urine was normal. We interpret that to mean normal for everything except bile. However, there is no blood or pus that would indicate hypernephroma.

We have considered syphilis as we are told definitely that this man has blood syphilis, or at least a positive Wassermann. I think it was Dr. Osler who said, "Know thou syphilis in all of its manifestations and all other things clinical shall be manifested unto you." We have taken Dr. Osler's axiom and have tried to make a case for syphilis. There is nothing in the picture that would indicate any form of hepatic syphilis. If we would consider hepatitis that would arise from an alcoholic source (the history tells us that he consumed only a moderate amount), we are in agreement that alcohol is no stranger to a man forty-six years old.

We do not know much about the liver. They do not tell us that they can feel it, that it is hard or nodular. They tell us only that they feel a mass in the region of the gall bladder. That fits very much better the picture of malignancy of an obstructive type.

Going back to syphilis, a recent investigation at the Mayo Clinic of a number of cases reveals that they were not able to differentaite the various types of syphilis of the liver from the pathological changes found in other forms of nonluetic liver involvements, such as the toxic, chemical, climatic, etc.

Except for the data given on the gall bladder, the liver seems to be left out of the picture. With a positive Wassermann, (as the Mayos have shown) there is a very magic response of the luetic hepatic conditions to mercury and iodides. We rather imagine if there was any suspicion of syphilis he would have been given anti-luetic treatment with a disappearance of symptoms. Instead, it is growing worse.

We must consider what would cause persistent painless jaundice. Gall stones could cause it but this condition is rare. We believe he would have had more of a colicky type of pain for a longer period, also that their attention would have been drawn to the possibility of occlusion by stone. From the history, we can not interpret anything in the past that would give him such a jaundice as he has without any other symptoms in the way of colic.

If we consider the various cirrhotic conditions, we are unable to differentiate them, as I have said before. To say positively that this is not visceral syphilis would be more or less speculative. But this type would have improved under treatment, and we believe that they would have given treatment.

This mass in the right side might be in the left lobe of the liver, but the history says it is a moveable mass about the size of an orange. In so many words, they say it feels like a gall bladder and believe it WAS a markedly dilated gall bladder. A condition in the kidney, described as hypernephrosis, could produce pressure and a water-logged kidney and could simulate this distention. There is no evidence of urinary disturbances and we believe that this mass would have been sufficiently large to call the patient's attention to it. We have no such evidence from the history.

In considering other conditions of the liver we have thought of hydatid cysts. But he has no tropical history of any kind. These tumors are usually large and again there is something impressive about them to the patient.

One sentence in the history, "A physician found fatty stools and told him his food was going through undigested." To us that means more than merely "his food was going through undigested." In pancreatic disease, an authority recently stated that a fatty stool is to be considered very seriously in the laboratory diagnosis of pencreatis. What condition in the pancreas would give us that picture of the gall bladder so dilated, associated with a definite mass in the epigastrium? We believe it was a malignant mass. Just what area it covered and just what metastases took place, we can not state, but we believe it was of sufficient size to involve the head of the pancreas, that it occluded the common duct and probably extended to the stomach; also perhaps it involved the retro-peritoneal structures.

To strengthen our impression of pancreatic malignancy a little further, we have looked up statistics and find that the French school of medicine has evolved a law known as "Courvoisier's Law," which states, in part, that when the common bile-duct is obstructed by a stone, dilatation of the gall-bladder is rare; when the duct is obstructed in some other way, especially by malignancy of the head of the pancreas, dilatation of the gall bladder is common. We rather believe that anything in medicine so dogmatic as to be incorporated into a law certainly merits attention.

This picture is rather vague as to the lung picture. The findings are rather misleading as to the terms employed or the interpretation of their findings is not exactly clear to us. They say "right upper chest dull, breath sounds loud and exaggerated throughout, no moisture." We are not able to interpret those findings otherwise than an indication of an existing fibrosis which would fit our own picture of fibrosis. He may have an old quiescent, latent tuberculosis which has given him no trouble and autopsy may show enough fibrosis to account for these findings. The possibility of pulmonary metastases has been considered, but we do not believe that they would have submitted this patient to an operation if they had thought that there was any possibility of malignancy in the lungs. The chest picture is rather confused.

Going back again to our speculation as to what operation was done,—if you remember the history, they were a little bit confused as to the diagnosis. He was told that he would live six weeks; and again he was 'privately informed" that they were not sure of the diagnosis even after the operatoin, and to go ahead and eat what he wanted. Yet they thought x-ray therapy advisable. He did eat what he wanted and got along fairly well for a time, and then all his symptoms returned.

Further speculating as to the second operation,—we rather believe that it was merely exploratory. Probably this malignant mass at the head of the pancreas was seen and noted so definitely that the man was simply closed up and nothing further in the way of surgery was considered advisable.

We believe that, while there were no urinary findings, this pitting edema of the extremities is associated with fluid in the abdomen. Ascites may not have been apparent. There is a discrepancy in the history. It was stated early that the abdomen was distended but nothing is mentioned of this distention in clinical examination data. We believe that the ascites could have been accounted for by pressure on the vena cava.

DR. JOHN W. FLINN, Prescott, Ariz.

In attempting very brief resume of this case, there are three or four outstanding facts that present themselves. The first is a man past forty years of age suddenly develops gastro-intestinal symptoms for the first time. This is always, we are told, strongly suggestive of gastro-intestinal cancer. Within the past week one of our group members has seen a case of very probable gastro-intestinal carcinoma in a woman fifty-eight years of age, who could mention practically the very hour of the very day in which she had her first gastro-intestinal symptoms, about four months ago. Another member of the group had a similar experience several years ago.

The second outstanding fact,—An afebrile persistent jaundice. The causes are three, first, gall stones and their effects; second, cancer of the pancreas or bile ducts, and third, cirrhosis. As was pointed out by the last speaker, Courvoisier's Law probably rules out gall stones in this case because we have jaundice and what we take to be an enlarged gall bladder, which, according to this law, are not both at the same time associated with gall stones.

Cirhosis is a possibility and, as has been pointed

out, it brings to mind the fact that there are serological findings of syphilis in this case. On the other hand, there are no definite findings of cirrhosis of the liver.

Practically all the classical symptoms of carcinoma of the pancreas are present in this case,—cachexia, pain, tumor in the epigastrium, probably enlarged gall bladder, fatty stools, gastro-intestinal disturbances, and edema of the legs and feet. The question is whether the cancer in the pancreas is primary or secondary. After considering the matter at great length we have come to the conclusion that it is quite impossible to tell. It is possible that the malignancy began in the pylorus and metastasized to the pancreas, but the fact that jaundice came on very early and the fact that there was very little vomiting point to primary carcinoma in the pancreas.

Our diagnoses are:

Carcinoma of the head of the pancreas,

Metastases in the liver and retro-peritoneal glands,

Hydrops of the gall bladder,

Edema of the lungs,

Probably a small ascites.

Discussions in the conference at the Massachusetts General Hospital were, in part, as follows:

RICHARD M. CABOT, M.D.

I am supposing that his jaundice ended after the six-weeks period.

I think if I were as uncertain as to diagnosis as they seem to have been in that first hospital I should say nothing about the prognosis. There is nothing that patients remember so vividly as having been told they are going to die in a few weeks and then getting better.

X-ray therapy ordinarily means malignant disease, of which we are not informed. I do not know how long he has had jaundice.

Dr. Tracy B. Mallory: He had it the whole time.

Dr. Cabot: Practically a year. That is very important. Chronic painless jaundice then is what we know, and it is all we know, up to the physical examination. Of course a year's jaundice is a pretty rare thing. We do not expect jaundice lasting a year from any of the common causes except malignant disease, and we do not expect it to last so long if it is malignant disease. So it is a particularly intriguing type of jaundice.

DIFFERENTIAL DIAGNOSIS

He died of a malignant disease, I should say, without any possible doubt. The only question is, where is it? We have not had an x-ray of his stomach. He has a good many stomach symptoms. It might perfectly well have been there, although it must, of course, have been outside as well in order to produce jaundice. It might have been in the head of the pancreas. Those are the two common places, although it mught have been in the gall bladder or bile ducts. I suspect if there are two tumors one was the distended gall bladder. We have nothing to prove cancer in the liver, although it may well be there. These signs in the lung suggest that there may be metastatic malignant disease there, although if it was as extensive as this it seems as if there would be other evidence of malignant disease.

FIRST OPERATION, MARCH 28, NINE MONTHS BEFORE SECOND ADMISSION

High right rectus muscle splitting incision. Exploration showed a very large and dilated gall bladder and dilated ducts fully an inch and a half in diameter, with a very hard head of the pancreas and enlarged lymph nodes in this area. There was no stone. It was decided that cholecystenterostomy would be of no benefit. The wound was closed without drainage.

PRE-OPERATIVE DIAGNOSIS JANUARY 14

Carcinoma of the pancreas.

SECOND OPERATION

Under local novocain a right rectus incision was made. A very large gall bladder presented, about seven inches long and four inches in diameter. This was seen to contain clear fluid. There was a hard mass in the region of the head of the pancreas with glands extending upward in the region of the common duct. The body of the pancreas felt cystic, as if the duct were dilated. The pyloric end of the stomach was negative. On aspiration the fluid was found to be slightly bile-tinged. On this account it was thought that the cystic duct was plugged and htat the condition of the gall bladder was hydrops. A cholecystgastrostomy was therefore not done. The perforation in the gall bladder was closed with two purse string sutures. The wound was closed without drainage.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Carcinoma of the pancreas.

DR. CABOT'S DIAGNOSIS

Cancer of the pancreas with metastases in the lung and the mesenteric glands.

Distended gall bladder.

ANATOMIC DIAGNOSIS

1. Primary disease.

   Carcinoma of the pancreas with liver and retro-peritoneal metastases.

2. Secondary or terminal lesions.

   Acute caseous pneumonia.

   Generalized miliary tuberculosis.

DR. TRACY B. MALLORY

I think the most interesting feature of this case is that a man showing as extensive pathology as we found could have been in such relatively good physical condition that the medical and surgical men who saw him felt that it was worth while to attempt a cholecystenterostomy to relieve his jaundice. That of course was the only possible operation that would have been of benefit to him. Up to the time of entry to the hospital he did not seem in any immediate danger of death. He had, however, a large carcinoma of the pancreas with immense metastases to the liver, the latter weighing 3000 grams, approximately double normal. He had also one entire lobe of his lung, the right upper, completely solid with acute caseous pneumonia, with a generalized miliary tuberculosis.

The reason that a cholecystenterostomy was not done was because the fluid in the gall bladder was colorless, and they presumed from that that the cystic duct was closed off and they would not be able to drain the liver through the gall bladder.

CASE OF ONE YEAR'S COUGH AND WEAKNESS

(Case No. 15011, Case Records of Massachusetts General Hospital, from N. E. Jour. of Med., Jan. 3, 1929, p. 31.)

CASE HISTORY

An American dairyman fifty-one years old entered August 6 complaining of weakness and fatigue of nine months' duration.

He was well until January, a year and nine months before admission. Then his house and farm buildings burned, ruining his business. Up to the next autumn he did not feel so well as usual, though he had weakness of the legs and malaise. He called a physician, who noticed that he was short of breath. He stopped all work. In January he had a severe nosebleed and two days later a second and a third milder ones. He had been subject to nosebleeds all his life, but not oftener than once a month and never very severe. His physician cauterized the nasal mucosa. Since that time there had been no more bleeding. His weakness, fatigue and dyspnea increased. For three weeks his appetite had been very poor. For two weeks he had been in bed and had had a few night sweats. He had lost thirty or forty pounds in the past year. He had possibly had some fever, but was not sure. For a year he had had very mild cough, not productive.

His mother died of cerebral hemorrhage and possibly had nephritis. His father had many nosebleeds. There is no family history of hemophilia. In ten years of marriage his wife had not been pregnant. She had one son by a previous marriage.

His habits were good.

At four years old he was in bed eighteen weeks with "rheumatism" in the left hip, leaving the hip stiff. Fifteen years before admission he had a rib broken. For fifteen years he had had painful hemorrhoids. Some years ago these bled. Some years before admission he had many sick headaches, at first one or two a month, later as often as once a week, keeping him in bed one or two days. The aching was always over the left eye. They were not relieved by glasses, but stopped when he stopped drinking coffee three years before admission. He had some loss of hearing. Recently he had had a few head colds. He had some abscessed teeth. A few years before admission his wife thought he was slightly dyspneic on exertion. He thought not. His memory was not so good as formerly.

The patient sometimes was very slow in replying, almost lethargic. Three times during the history-taking he went to sleep. The history of the present illness was not considered complete.

Clinical examination showed a pale, slightly dyspneic, undernourished man. Over the trunk and upper arms were scattered round brownish macules 5 to 10 millimeters in diameter with fairly definite borders and thin atrophic or scaling centers. Marked caries and pyorrhea. Perforated nasal septum. Very slight excursion of the diaphragm on deep inspiration, probably not over one centimeter. Respiration very shallow. No definite change in percussion note made out. Fine and coarse rales at both bases, front and back, higher on the right. Location of the apex impulse of the heart not recorded. No enlargement to percussion. Action regular. Sounds of fair quality. A systolic murmur at the base and at the pulmonic and aortic areas. No diastolic heard. Arteries normal. Blood pressure 98/46. Spleen felt below the costal border on inspiration. Generalized moderate atrophy of the left leg, which was shorter than the right. Left hip joint apparently ankylosed. Questionable slight motility in flexion. Protruding internal hemorrhoids, not painful. In the posterior anal margin was a fistulous opening in the skin about two millimeters in diameter. Pupils normal. Right knee-jerk very sluggish on reenforcement. Left knee-jerk active. Ankle-jerks not obtained. Cremasterics and abdominal reflexes normal.

Urine 28 to 70 ounces, specific gravity 1.010 to 1.016, a very slight trace of albumin at the last of four examinations, an occasional leukocyte once. Blood: 8,750 to 15,700 leukocytes, 79 to 83 per cent polymorphonuclears, hemoglobin 60 to 55 per cent, reds 3,240,000 to 4,350,000, smear normal. Hinton negative.

Sputum not remarkable on five examinations; no blood or unusual organisms. Sputum culture showed a yeast-like organism in almost pure culture. Stools negative. Fasting contents of stomach: 40 cubic centimeters of cloudy colorless fluid, free acid 5, combined acid 16. Test meal: 30 cubic centimeters of similar fluid, free acid 29, combined acid 23. Both specimens gave negative guaiac and on microscopic examination showed 10 to 12 polymorphonuclears per high power field.

X-ray examination August 8 showed the diaphragm low. The respiratory movements were limited on the right, fairly good on the left. The costophrenic sinus was obliterated on the right. There was increased density throughout the greater part of both lung fields. The shadows were coarse and mottled. There were many dense white lines running through them. In the midpart of the right lung field there was a bright area surrounded by rather sharply defined margins of increased density, somewhat suggestive of cavity formation. No fluid level was present. There were no mediastinal masses. "I do not believe that this is tuberculosis." The heart was not remarkable. There was marked deformity of the head and neck of the left femur. The joint space was not obliterated. There was no marked deformity or erosion of the joint surface. The sinuses were negative. There were numerous carious teeth and apical abscesses and marked alveolar retraction. August 17 a second examination of the chest showed no appreciable change since the first observation.

Temperature 98.2° to 104.5°, with evening rise. Pulse 71 to 125. Respirations 20 to 37.

The patient showed striking dyspnea and weakness. Dullness seemed to extend to the right of the sternum without obvious cardiac enlargement. August 8 the rales were considered fine crepitant. There was questionable dullness at both bases. A skin consultant made a diagnosis of possible tinea versicolor. An orthopedic consultant reported, "The hip is the result of an old slipped epiphysis. No tuberculosis." A surgical consultant reported, "Posterior fistula in ano, very punched out opening; suggests tuberculosis."

The patient grew gradually weaker. He had no more hemoptysis (sic). The visiting physician thought the lung process became more extensive. August 25 the patient very suddenly died.

DISCUSSIONS.

The following discussions of this case were given by Group 3, of the Yavapai County Medical Society and Medical Officers of Fort Whipple, at their joint meeting of Feb. 25, 1930:

DR. G. O. BASSETT, Whipple, Ariz.

This case is filled with rather peculiar facts. One of the most peculiar is that he did not lose his appetite sooner. He did not lose it until three weeks before admission. Had my house and barns burned down, my appetite would have disappeared immediately.

His illness dates back about eighteen months. He gives a picture of a chronic lingering illness. He lost thirty to forty pounds in a year. He is cachectic, he is weak, he has a very definite lowering of blood pressure, systolic being less than 100. The first assumption would be that we have a case of tuberculosis. He gives a history of nosebleed, which was checked by treatment, and which apparently had nothing to do with the picture. It may very easily be a case of pulmonary tuberculosis. He is certainly ailing for a period of eighteen months; he has a cough, but very slight expectoration. Occasionally we see tuberculosis with that feature,—persistent cough and very little or no expectoration. He is increasingly dyspneic, probably in proportion to the spread of disease in the lungs. Whether there are complications, we do not know.

Unfortunately five sputum tests are made and nothing is found; not only do they not find tubercle bacilli, but nothing else is found. Also an x-ray is taken and reported not tuberculosis. That x-ray report of the lung is very interesting. There is coarse, diffuse mottling, gross, heavy fibrous bands from the periphery to the hilus. probable cavity formation in the right side. If this were tuberculosis, with that amount of involvement and the cough present, we would expect to find something in the sputum. The people who examined the x-ray and the laboratory people who examined the sputum both agree that this case is not tuberculosis, so we are compelled to rule it out.

Among other chronic illnesses we have to consider malignancy of the lung. Shultz, I believe it was, said that malignancy of the lung is not rare, and to confirm his statement gave a review of one hundred thousand autopsies. of which one hundred and fifty-six were found to have pulmonary malignancy. Primary malignancies of the lung are rather rare, metastatic tumors being about ten to one. Both give pretty much the same picture. There is massive infiltration on one side or the other, or possibly both. The general picture this man presents could very easily fit into that of an individual suffering with malignant disease of the lung. The x-ray is not negative. Carcinoma sometimes develops cavities. Unfortunately, there is no record of malignancy of the lung, where it causes death. which does not show blood in the sputum, hemorrhages and usually very foul sputum. He coughs but does not expectorate. He has no record of any bleeding, although it states in the last paragraph of the history, after ignoring the fact that he had no sputum, it states he had no *further* hemorrhages.

What kind of malignancy could this be without those symptoms? There is wide-spread mottling. There is a type of malignancy of th elung which takes on more of an infiltrative character, spreads to the hilus and all parts of the lungs, and gives a scattered. generalized mottling. So it is impossible to rule out malignancy of the lung, somewhat out of the ordinary, and metastasized from a primary growth of which we can find no evidence. There is nothing in the abdomen; no record of anything about the prostate.

There is a type of malignancy of the lung which have to consider. This is the blastomycotic infections, which our case would fit. However, as in tuberculosis and malignancy, we find there are conditions which do not quite meet the problem. These conditions resemble tuberculosis so closely, as far as x-ray is concerned, that it is impossible to differentiate the type of involvement. Thickened pleura is typical. The clinical findings and objective symptoms are very much the same.——cough, pleurisy and pains. However, with the profuse sputum that these cases usually have, it is not difficult to make the diagnosis. the blastomyces being present in the sputum. They examined this man's sputum and tell us not only that it is not tuberculous but that no other organisms were found. These fungus infections are characterized by a definite ulceration at some point or other, where the infection enters and spreads to the lung or other parts of the body, neck. head, face, etc.——almost invariably multiple areas of ulceration are found.

One other disease we mention simply to rule out,—syphilis. We take the laboratory findings for what they are worth,—there is a negative Hinton.

That brings us to one statement in the history. They say that five sputum tests are not only negative for tuberculosis but for everything else. Then they mention a pure culture of a yeast-like organism, so that after all the sputum is not negative.

DR. B. C. JONES, Whipple, Ariz.

This case presents symptoms of cancer, of mycotic infection and of tuberculosis. One method of determining whether it is tuberculosis or mycotic infection is by laboratory assistance. In a case which runs along like tuberculosis and the organism of tuberculosis is not found, it should be determined whether or not it is due to one of the mycotic organisms. It is believed at the present time that it occurs many more times than we think.

In this case fove sputum tests were negative for tuberculosis and then a yeast organism was found. To find a yeast organism in the sputum does not necessarily mean that the disease of the lungs is of that infection, but it is up to the investigator to determine what type of organism it is. whether it is a pathological form, whether it is saprophitic, or whether it is the definite cause of the disease present.

C stellani gives under bronchomycoses the following yeast-like organisms: Broncho-moniliasis, blastomycosis, cryptococcosis, saccharomycetosis, bronchoendomycosis.

I think it is impossible for us to determine what organism is present. We can only go so far as to say it is one of the mycoses. The small amount of sputum and the duration of the disease, a year and a half, points to myotic infection and also to malignancy of the lung. In a report of a large series of primary cancers of the lungs, the duration ranged from two years to a few months from the time of discovery.

In mycotic infection of the lung there is usually cough. scant expectoration, loss of weight and blood in the sputum. The same is true of cancer of the lung,—dyspnea, cough, loss of weight, hemoptysis may occur, fever not marked, and the duration about the same. I shall not go into the different types of mycotic infections because it is practically up to the laboratory to determine the organism present.

In this case the history mentions something about the heart and something about rheumatism in the early part of his life, and then about the patient going to sleep several times during examination, which it is thought may possibly be a heart infection, embolism. or some brain lesion. We have not put great stress on this heart. If this man has chronic disease of the heart, we certainly do not expect it from the report of examination. We do not put much stress on the murmurs which they state are present, and think that low blood pressure is due simply to low vitality.

To make a statement about the terminal condition would be just speculation. A man of fifty-one years dies suddenly. Many things could happen, possibly terminal pneumonia. The progress of the infection is in the lungs.

Our diagnosis is mycotic infection of the lungs.

The following discussions of the case were

given at the Monday noon meeting of the Phoenix Clinical Club on June 20, 1930:

DR. J. M. GREER

The history states that this man was fifty-one years of age, a dairyman and well, in so far as he knew, up to a year and nine months ago. Given this history we immediately think of some more or less chronic disease and one consistent with an active outdoor life rather than one associated with a sedentary life. That his farm buildings burned may be of some importance in the history, in that the disease from which he is suffering may be of much longer standing and the fire precipitated the symptoms or brought them more forcibly to his attention.

The shortness suggests to us, pulmonary disease, disturbance of the cardio-vascular system or disease of the cardio-vascular-renal system. The history of nose bleed suggests that we must be on the lookout for arteriosclerosis or some local condition of the nasal mucous membrane. However we must not forget that many people have nose bleed without appreciable disease of the vascular system. In this case the nose bleed was probably due to a hypertrophied nasal mucosa or varicosed and tortuous nose vessels. as it promptly discontinued upon cauterization.

The weakness, fatigue, dyspnea with some night sweats, as well as the loss of weight and cough, bring our attention back to the chest.

The family and marital history do not mean much to me.

The eighteen weeks in bed at the age of four with "rheumatism" followed by a stiff hip means tuberculosis, unless it can be absolutely ruled out by x-ray or other means. The hemorrhoids may mean a passive congestion from some general condition. This passive congestion is probably the cause of the enlarged spleen. The sick headaches may mean a gall bladder or liver disturbance, but as these cleared up upon discontinuance of coffee they were probably an intolerance to the caffeine or some other constituent of the coffee.

The reported lethargic condition of the patient could be accounted for by a toxemia. However, the history does not point toward any great degree of toxemia.

Upon physical examination the pallor, dyspnea and undernourishment suggests some chronic pulmonary affection.

The skin findings indicate some independent skin disease and we note farther on it was diagnosed as "tinea versicolor." No doubt if Dr. Clohessy could see this he could immediately prove or disprove this diagnosis. The point I am trying to make is that some dermatologist would have to see the lesion in order to make a diagnosis. I do not believe that it had anything to do with the patient's general condition.

The perforated septum suggests lues, but it is the only thing that does. And we know that other things may account for a perforated septum, as in this case the repeated nose bleeds and the, no doubt, repeated trauma, as well as the cauterization as noted in the history. These factors may have been responsible for the perforated septum.

The physical findings of the chest point toward some lung affection. The systolic murmur heard over the base of the heart could mean an old valvular lesion or probably might have been one of the so-called functional murmurs. The low blood pressure is probably due to the generally weakened condition of the patient. Possibly there was some myocardial degeneration present.

It is noted that the sputum is not remarkable. I do not know just what is meant by this. I do not know just what a remarkable sputum would be either. The finding of the yeast-like organisms on culture of the sputum is very important and to my mind gives us the major diagnosis. The x-ray findings of the hip practically rule out a tuberculous arthritis.

Diagnosis:—
Fungus infection of the lungs.
Myocarditis and endocarditis.

The sudden cause of death was probably due to acute dilatation or embolism.

DR HOWELL RANDOLPH

Gradually increasing weakness for a year and nine months, loss of 40 pounds in weight during the past year, mild cough, dyspnea, center attention upon the chest, and of course the most common disease of the chest causing this group of symptoms is tuberculosis. Many other points in the record tend to substantiate such a diagnosis. However. there are certain findings which bring in the question of some other etiological agent.

This patient's occupation was that of a dairyman. Working about animals and in the open,—like farmers and bakers, etc.,—is somewhat more apt to subject a person to fungus infections especially actinomycosis. There were yeast forms found in the sputum in almost pure culture, tho not observed in five different sputum smears. Yeast forms are not infrequently found in the mouth, and for this reason, it is necessary to brush teeth and rinse mouth thoroughly before collecting the specimen. This man had marked oral sepsis. However, the finding of almost pure culture in this case is very significant. Howe and Schmidt in reporting ten cases of yeast infection, state that "the finding of two or three colonies on a plate be considered of pathological significance," whereas they pay little attention to finding of single colonies.

X-ray findings seem rather indefinite. Coarse mottling in the x-ray film is consistent with fungus infection, miliary carcinosis or tuberculosis, pneumoconiosis. The man's occupation had not exposed him to dust to any extent or it would have been mentioned.

Of the fungus infections, actinomycosis is not suggested by x-ray description. As a rule this fungus causes dense foci in the hilus region with streaks or radiation from the central density. Streptothrix is very closely allied to actinomycosis.

Blastomycosis is one of the more common fungus invaders of the lung. However, the x-ray findings are apt to be more suggestive and the course of the disease somewhat more rapid. In favor of carcinoma with metastasis is the age and the finding of gastric contents of slightly lowered acidity and leucocytes. But continued high fever practically rules malignancy out. Miliary tuberculosis can very well explain the clinical picture. The hip joint disease may well have been tuberculosis in spite of the opinion offered. It is quite likely that the miliary spread may have taken place two or three months before admission repeated before death. The negative sputum tests and the x-ray report are against miliary tuberculosis together with the absence of symptoms referring to gastro-intestinal tract.

Perforation of septum was probably traumatic. Systolic murmur is most likely due to anemia which is rather severe in this case. The difference in reflexes is secondary to changes in the hip.

The fistula in ano seems to me to be a very important differential point in this record and when taken with the other findings limits the diagnosis to tuberculosis and fungus infection.

Diagnosis. Fungus infection of lungs.

Comment. Areo Leao has studied the reactions to filtrates from cultures of actinomycosis and has developed a preparation which reacts positively in clinical cases of the disease and negatively in normal individuals. He also found agglutination test positive in 1:160 dilution. The preicipitin test was

negative. The complement fixation test was positive.

Diagnostic conclusions on this case were offered as follows:—

Dr. H. P. Mills:—Tuberculosis.

Dr. Fred Holmes:—Pulmonary tuberculosis; old tuberculosis of hip; rectal fistula; perhaps fungus infection secondary.

Dr. T. T. Clohessy:—Pulmonary blastomycosis.

Dr. F. C. Jordan:—Tuberculosis.

Dr. Victor Randolph:—Pulmonary tuberculosis; old tuberculous hip; secondary fungus infection of lungs.

Dr. F. J. Milloy:—Tuberculosis.

Dr. O. H. Brown:—Blastomycosis; syphilis (?); myocarditis; endocarditis; tinea versicolor; secondary anemia.

Dr. R. J. Stroud:—(1) Some fungus disease of lungs; (2) tuberculosis of lung.

Dr. W. W. Watkins: History suggests lung disease, heart disease or primary anemia; clinical examination suggests heart disease, lung disease, primary anemia or syphilis; laboratory examinations rule out anemia and heart disease; establishes lung disease, miliary tuberculosis, fungus lung disease or malignancy. I favor fungus disease of lungs as diagnosis.

The following are the discussions before the Clinical Conference at the Massachusetts General Hospital:—

DR. RICHARD C. CABOT,

It seems as if the family history was important here. We have something suggesting arterial disease in the patient, also in both of his parents.

I suppose he had tuberculosis of the hip.

We used to say that perforated nasal septum means syphilis. Now they say it may mean syphilis but may mean any other pyogenic infection of the same locality.

"Ankle-jerks not obtained," in connection with his perforated septum, makes us wonder as to tabes.

What have we so far? We have a secondary anemia apparently, certainly an anemia. We have an old hip disease which certainly has nothing to do with his present trouble, a rather low blood pressure, a perforated nasal septum, some queer reflexes, and a skin trouble that I do not believe has anything to do with his death. All of that put together is not enough to account for his having died.

I think even the uninitiated like ourselves would say that there is a good deal wrong with those lungs as shown in the x-ray plates. There is a tremendous amount of shadow well scattered throughout both lungs. It could not possibly be normal. It must be pathological.

Let us speculate a little on the grounds of the radiologist's opinion that it is not tuberculosis. In the first place, there is less trouble at the top than at the base. That is almost enough to exclude tuberculosis. We shall come back later to what we have.

Tinea versicolor of course has no connection with this case.

In the final paragraph is the first mention we have had of hemoptysis.

DIFFERENTIAL DIAGNOSIS

I do not believe I know what he has. Let us go through the things that we know in the lungs. Of course tuberculosis is the first thought. Five examinations of the sputum showed no tubercle bacilli. Chronic bronchopneumonia, non-tuberculous, is a real thing, from time to time brought up in reports. I do not know how anybody is going to say this is not a chronic non-tuberculous bronchopneumonia. Syphilis of the lung I have already said we cannot say anything about. There is no fresh material or necropsied case on which to base any clinical picture of syphilis of the lungs. Pneumoconiosis: it does not seem as if he could have been exposed to the type of irritant that gives rise to that. I do not think that with this history we can say that. I think I have seen x-ray pictures of pneumoconiosis that looked not unlike this. I cannot think of anything else in this climate.

A Student: Fungus infection?

Dr. Cabot: That is possible, but we have no evidence. They had a good chance to look at his sputum.

A Student: It says "yeast." Could it be blastomycosis?

Dr. Cabot: Blastomycosis of the lungs is due to a wholly different organism from this. But I will admit that I know very little about yeast organisms in the lungs. If that is a possibility I do not know enough to say anything about this one way or another.

A Student: How about actinomycosis? He was a dairyman and the sputum showed a yeast-like organism.

Dr. Cabot: That could be recognized only by sputum examination. I should say they had a good chance to look at the sputum and they did not find actinomycosis. If a yeast-like organism can do this thing, then there is nothing more to be said. If so I do not know it.

A Student: Is the lung picture consistent with metastatic carcinoma?

Dr. Cabot: I should not suppose it was. If it is not a non-tuberculous bronchopneumonia, which is a rather unsatisfactory diagnosis, I do not know what it is.

INTERFRETATIONS OF X-RAYS

The appearance is that of an extensive diffuse fibrosing process in both lungs with involvement of the pleura on the right.

The picture of the left hip joint is not characteristic of an old tuberculosis, but could be due to any destructive lesion.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Yeast infection of the lung.

DR. RICHARD C. CABOT'S DIAGNOSIS

Chronic non-tuberculous bronchopneumonia.

DR. TRACY B. MALLORY

ANATOMIC DIAGNOSES

1. Primary disease.
   Actinomycosis of lungs.
2. Secondary or terminal lesions.
   Passive congestion and central necrosis of liver.
   Necrosis in adrenal.

Dr. Cabot's diagnosis is correct. but the bronchopneumonia was due to the actinomyces. The lung was filled with small cavities and also many small fibrous tubercle-like nodular granulomata which grossly greatly resembled tubercles. The largest of the cavities was about one and a half centimeters in diameter. Many of them contained small amounts of pus, others were rather dry, and the content rather suggestive of caseous material. There was no spread of the lesion to the other organs. Metastatic lesions are of course not frequent in actinomycosis.

NOTES BY DR. CABOT

1. It is remarkable how much more trouble the x-rays showed than could have been expected from the record of the chest examination. It emphasizes the necessity of x-ray examination in all patients with chronic cough.

2. Dr. William H. Smith seems to be the only person in this vicinity who can be trusted to recognize actinomycosis from sputum examination.

CASE OF DYSPNEA AND COUGH

(Case 152818, Case Records of Massachusetts General Hospital. from N. E. Jour. of Med., July 11, 1929, p. 92.)

CASE HISTORY

A married Canadian woman thirty-seven years old entered January 21. moribund. The chief complaint was cough and dyspnea. A hurried and incomplete history was obtained from the patient and her husband.

Three years before admission she was ill in bed for three weeks with influenza with fever. She had a prolonged convalescence; no inflamed joints. Since that time she had been somewhat limited in her activities by dyspnea, slept on two pillows and had transient evening edema of the ankles. She got along fairly well, however. until three weeks before admission, when she had a slight cold with sore throat, fever and malaise. She developed a persistent cough with yellow and occasionally blood tinged sputum. She had constant pain in the right upper quadrant aggravated by coughing. The dyspnea became worse. She had slight swelling of the ankles. In spite of medical treatment she went downhill steadily.

Clinical examination showed a poorly developed and nourished, very pale woman, moderately cyanotic in artificial light, in acute respiratory distress. Teeth poor. Much dentistry. Throat injected. Tonsils moderately enlarged. Loud coarse moist rales throughout the chest with distant breath sounds at the bases but no fluid. Apex impulse of the heart diffuse and heaving, seen and felt in the 5th space 10 to 11 centimeters from midsternum. Sounds and action normal. Left border of dullness 13 cm. to left of midsternum, 6.5 cm. outside the midclavicular line. Right vorner of dullness 4.5 cm. Heart of the mitral shape. A loud blowing apical systolic murmur. No diastolic heard. (Extraneous sounds made the examination unsatisfactory.) A questionable apical systolic thrill. Pulmonic second sound accentuated. Pulses and arteries normal. Blood pressure 110/90. Liver enlarged to below the unbilicus. tender and painful. Slight edema of the ankles.

Temperature 100.4° to 102°. Pulse 120. Respirations 40 to 45. Leukocyte count 20,000. .

Urine not recorded.

The patient looked very anemic. Venesection was considered, but seemed inadvisable. She was given intramuscular digifolin, also morphia and caffein. She slowly failed and six hours after admission died.

Discussed by the Phoenix Clinical Club, at their regular Monday noon luncheon. Diagnostic conclusions by indiidual members of the Club were given, as follows:

DR. F. C. JORDAN:—Rheumatic endocarditis; abscess or malignancy of liver.

DR. T. T. CLOHESSY: Endocarditis; mitral insufficiency; pulmonary and heptic passive congestion; pneumonia.

DR.. H. P. MILLS:—Endocarditis; pulmonary edema.

DR. F. J. MILLOY:—Bronchopneumonia; cardiac decompensation caused by mitral regurgitation.

DR. W. W. WATKINS:—Mitral valve disease.

DR. V. RANDOLPH:—Chronic endocarditis involving mitral and possibly tricuspid valves; pulmonary infarct on right; possibly termnial pneumonia; chronic nephritis.

DR. FRED HOLMES:—Chronic ondocarditis of mitral valve.

DISCUSSION BY DR. ORVILLE H. BROWN

The salient points in this record are: married woman, 37 years of age. moribund on entering hospital; cough and dyspnea chief complaint. Three years before, she had influenza; slow convalescence and ever since dyspnea. Respiratory tract infection developed three weeks before admission producing purulent blood tinged sputum. She had constant pain in the right upper quadrant exaggerated by cough.

Examination: Emaciated, pale, cyanotic woman. Throat inflamed; tonsils enlarged; coarse rales throughout chest; indistinct breath sounds at bases. Heart enlarged, loud systolic murmure. Liver enlarged. Edema of ankles. Leukocytes 20,000; respiration 45; pulse 120; temperature 102. Given digitalis, morphine and cammeine. Six hours after admission to hospital she died.

This is the type of inadequate record so often obtained in acutely ill cases. It leaves much to be desired and much to be guessed at.

The sequence of events seems to have been an infectious process three years before admission which had a profound effect upon her. She had a slow convalescence and ever afterwards had dyspnea. One should think of tuberculosis as being the cause of the slow convalescence and possibly even of the dyspnea. Nothing is said, however, about a protracted cough after the influenza, nor is anything said of sputum. fever and other usual symptoms of tuberculosis. .

Nothing is said of asthmatic breathing; therefore, we must assume that dyspnea was not of the asthmatic type. There was a small amount of edema in the ankles, but there was no other finding to suggest kidney complication or disease. The rales spoken of might be interpreted as indicating chronic bronchitis and an asthmatic tendency. I have, on several occasions, had patients who found it difficult to describe their asthmatic attacks so one really felt sure that they knew what they were talking about. .

Tumors or growths about the chest or mediastinum may produce dyspnea, but nothing is in the record to suggest these. In severe anemia there is usually dyspnea, but it would hardly seem warranted to assume that dyspnea in this case came from anemia.

There is a discrepancy in the record about the size of the heart and heart sounds. I interpret the records as of two separate examinations. It seems certain that there was a dilated heart at the time of admission to the hospital and that this became worse. At the time of admission the sounds and action of the heart seemed normal, except that a heaving impulse was felt and seen. Later there was a thrill. At this time the heart was found further loud blowing apical murmur with a questionable dilated and it seems reasonable to assume that the murmur and thrill (?) were the result of dilation.

The liver was enlarged and there was constant pain and tenderness in the right upper quadrant. Both of these findings suggest the damming back of the blood from the right heart. A filling of the lungs with mucous rales and an accentuated second pulmonic suggest damming back of blood from the left heart to the right. The pulmonary moisture suggests pulmonary edema. The constant pain in the right upper quadrant might be more than a congested liver. but there is nothing in the record to suggest gall stones or infected gall bladder, as these are common findings one might guess that such existed. The enlarged liver might make one think of a malignancy, but there is nothing else in the record to suggest malignancy. An enlarged liver may arise from a variety of causes such as: abscess. diabetes, carcinoma, catarrhal jaundice. cirrhosis, syphilis, etc. There is no finding to substantiate the diagnosis other than prolonged passive congestion.

The condition of the heart might be a pericarditis. I think, however, there would have been more syanosis. There might well have been endocarditis.

My guess at the diagnosis is:

1. Death due to acute cardiac dilatation.
2. Pulmonary edema.
3. Chronic cardiac dilatation; moderate decompensation.
4. Chronic tonsillitis.
5. Enlarged liver from passive congestion.
6. Possibly, pulmonary tuberculosis.

Discussion at the Clinical Conference at the Massachusets General Hospital, in part was as follows:

DR. RICHARD C. CABOT

Here again we have to deal with a case without proper study until the end of life.

What they probably found at the bases was dullness, diminished tactile framitus and breath sounds. It is improper to put that down as "fluid." You do not hear fluid or see fluid.

The diffuse heaving impulse shows hypertrophy. The location of the apex, however. does not show enlargement and is against what the heaving impulse suggests. The midclavicular line ordinarily corresponds with the nipple, so 6.5 centimeters outside the nipple is a good deal. It seems as if there were considerable cardiac enlargement by percussion but not by palpation.

The "mitral shape" means that the heart shadow sticks out in the left upper border corresponding to the left auricle. As the examination was unsatisfactory very likely there was a diastolic murmur.

Notice that the pulse pressure was only 20, very low.

DIFFERENTIAL DIAGNOSIS

I think we can say she died of heart disease. We have nothing to indicate any other kind of death. What type of heart disease did she have? She was pretty young to have hypertension. There is no suggestion of hypertension. no evidence of syphilis, no evidence of rheumatism. She is of the age and sex in which usually if there is heart disease it turns out to be rheumatic. Hence my guess is that it is rheumatic. If it is rheumatic heart disease what valve does it usually affect? I am guessing again; mitral stenosis, in spite of the fact that we have only a systolic murmur. If we had had a good chance we might have heard a diastolic murmur. That seems to be the best guess we can make. She was very pale. I am very anxious but unfortunately unable to answer the question, did she have anemia? If she had anemia the diagnosis I have made is not enough. We do not get any anemia from rheumatic heart disease. If she was really anemic I suspect subacute bacterial endocarditis on top of an old process or alone. We cannot do more than speculate about that. as we have no blood culture and could not follow up during life. If she had it, it would account for the leukocytosis.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Rheumatic heart disease with congestive failure.
Mitral stenosis and insufficiency.
Bronchopneumonia.

DR. RICHARD C. CABOT'S DIAGNOSIS

Heart disease, probably rheumatic endocarditis of the mitral valve (mitral stenosis).
Possibly subacute bacterial endocarditis.
Chronic passive congestion of all the organs.
Bronchitis.

DR. TRACY B. MALLORY

ANATOMIC DIAGNOSIS

1. *Primary disease.*
   Acute and chronic rheumatic endocarditis and pericarditis; mitral stenosis.
2. *Secondary or terminal lesions.*
   Acute bronchitis and slight bronchopneumonia.
   Chronic passive congestion.
   Slight arteriosclerosis.

The chief finding in the heart was a marked degree of mitral stenosis. There was acute endocarditis with fresh processes on the mitral, tricuspid and pulmonic valves and also both an acute and an old chronic pericarditis. Myocarditis was observed microscopically.

She had a slight degree of bronchitis but no pneumonia.

The liver and other organs showed marked congestion.

Dr. Cabot: Will you say something more about the myocarditis

Dr. Mallory: There were lesions of two types. One variety consisted of typical Aschoff bodies. collections of the large mononuclear cells of the tissues which are beginning to be called clasmatocytes nowadays, together with some fibroblasts, the typical pathology of rheumatic infection of the myocardium. There was also acute myocarditis with necrosis of muscle cells and invasion by polymorphonuclears. This acute myocarditis is in all probability not rheumatic in character but a superinfection. Cultures showed streptococcus, as is usual in these cases.

Dr. Cabot: I should like to emphasize certain points. (1) She had mitral stenosis, and we ought to have heard a diastolic murmur. (2) She was pale and presumably anemic, corresponding with the acute endocarditis which Dr. Mallory finds. If we had taken the hemoglobin we should have found a lowering. (3) The liver was enlarged from nothing but passive congestion, as I predicted.

THE JOHN PHILLIPS MEMORIAL PRIZE

The American College of Physicians announces the JOHN PHILLIPS MEMORIAL PRIZE of $1500, to be awarded for the most meritorious contribution in Internal Medicine and sciences contributing thereto, under the following conditions:

(1) The contribution must be submitted in the form of a thesis or dissertation based upon pubilshed or unpublished original work.
(2) It must be mailed to the Executive Secretary of the American College of Physicians on or before August 31, 1930.
(3) The thesis or dissertation must be in the English language, in triplicate, in typewritten or printed form, and the work upon which it is based must have been done in whole or in part in the United States or Canada.
(4) The recipient of the prize would be expected to read the essay at the next Annual Meeting of the College, after which he would be officially presented with the prize by the President.
(5) The College reserves the right to make no award of the prize if a sufficiently meritorious piece of work has not been received.
(6) The announcement of the Prize winner will be made not later than two months before the Annual Meeting.

AMERICAN COLLEGE OF PHYSICIANS,
E. R. Loveland, Ekecutive Secretary,
133-135 S. 36th Street,
Philadelphia, Pa.

NEW MEXICO ANNUAL MEETING

NOTES OF THE MEETING

The attendance was very good and as the weather was delightful, with the exception of the hard shower which came right after the adjournment sine die, the beautiful scenery around Raton was thoroughly enjoyed by all.

Those registering were:

Dr. E. N. Allan, Rochester, Minn.
Dr. W. T. H. Baker, Pueblo, Colo.
Dr. O. H. Brown, Phoenix, Arizona.
Dr. W. L. Brown, El Paso, Texas.
Dr. R. O. Brown, Santa Fe, N.M.
Dr. J. M. Britton, El Paso, Texas.
Dr. C. R. Bass, Cimarron, N. M.
Dr. L. B. Cohenour, Albuquerque, N. M.
Dr. C. H. Churchill, Madrid, N. M.
Dr. P. T. Cornish, Jr., Albuquerque, N.M.
Dr. F. E. Diemer, Denver, Colo.
Dr. E. Durham, Raton, N.M.
Dr. D. C. Daniel, Mosquero, N. M.
Dr. F. C. Diver, Dawson, N.M.
Dr. Crum Epler, Pueblo, Colo.
Dr. C. Ernest, Pueblo, Colo.
Dr. C. B. Elliott, Raton, N.M.
Dr. Tobias Espinosa, Espanola, N.M.
Dr. R. H. Finney, Pueblo, Colo.
Dr. J. E. Funk, Santa Rosa, N.M.
Dr. C. B. Francisca, Kansas City, Mo.
Dr. R. J. Groom, Santa Rita, N.M.
Dr. M. D. Gibbs, Roy, N.M.
Dr. A. C. Grimes, Halstead, Kas.
Dr. C. S. Hart, Dawson, N.M.
Dr. and Mrs. C. S. Harper, Swastika, N. M.
Dr. J. W. Hannett, Albuquerque, N. M.
Dr. John B. Hartwell, Colorado Springs, Colo.
Dr. L. A. Hubbard, Gardiner, N. M.
Dr. F. M. Heller, Pueblo, Colo.
Dr. Home J. Herymann, Raton, N. M.
Dr. W. T. Joyner, Roswell, N. M.
Dr. E. W. Johes, Fort Collins, Colo.
Dr. W. E. Kaser, Las Vegas, N. M.
Dr. W. M. Lancaster, Clovis, N. M.
Dr. J. R. Lemmon, Amarillo, Texas.
Dr. W. J. Latta, Wagon Mound, N. M.
Dr. T. B. Lyon, Raton, N. M.
Dr. H. A. La Moure, Pueblo, Colo.
Dr. Walter Martin, Battle Creek, Mich.
George A. McAlmon, El Paso, Texas.
J. H. MacWhorter, El Paso, Texas.
Dr. C. F. Milligan, Clayton, N. M.
Dr. S. M. Parrish, Raton, N. M.
Dr. Stuart Pritchard, Battle Creek, Mich.
Dr. J. F. Perry, Los Angeles, Cal.
Dr. and Mrs. J. P. Powell, Dalhart, Texas.
Dr. I. L. Peavy, Van Houten, N. M.
Dr. W. R. Quinn, Dawson, N. M.
Dr. P. E. Rice, Raton, N. M.
Dr. T. F. Self, Roy, N. M.
Dr. A. B. Stewart, Raton, N. M.
Dr. A. J. Streit, Amarillo, Texas.
Dr. M. F. Smith, Raton, N. M.
Dr. E. B. Shaw, Las Vegas, N. M.
Dr. D. Tellefson, Los Angeles, Cal.
Dr. L. A. Thompson, Springer, N. M.
Dr. F. D. Vickers, Deming, N. M.
Dr. J. R. Van Atta, Albuquerque, N. M.
Dr. S. L. Wilkinson, Belen, N. M.
Dr. M. K. Wylder, Albuquerque, N. M.
Dr. O. J. Whitcomb, Raton, N. M.

The office of President-Elect was contested in a friendly way by Dr. M. K. Wylder, Albuquerque, and Dr. F. D. Vickers, Deming, the former winning by an eye (aye) as it were. Dr. Vickers had demurred when his name was proposed, stating that the President should reside in a more central part of the state and since Albuquerque would probably be the next meeting place, it was only proper that the President should be a resident of that city.

Other officers elected were:

Vice-President—Dr. F. D. Vickers, Deming;

Secretary-Treasurer—Dr. L. B. Cohenour, Albuquerque, re-elected.

Members of the Council for three years—Dr. W. T. Joyner,Roswell, re-elected; Dr. H. A. Miller, Clovis, re-elected.

Member of the Council to serve the unexpired term of Dr. Dwight Allison, removed from state—Dr. C. W. Gerber, Las Cruces.

Delegate to A.M.A.—Dr. H. A. Miller, Clovis. Alternate—Dr. W. J. Latta, Wagon Mound.

Members of Board of Managers Southwestern Medicine—Dr. P. G. Cornish, Jr., Albuquerque, Dr. A. B. Stewart, Raton.

Albuquerque was chosen unanimously as the meeting place for 1931.

The banquet at the Hotel Swastika was well attended and thoroughly enjoyed by all. The ballroom of the hostelry is very beautiful, and when, as on this occasion, graced by the many charming ladies of Raton and elsewhere, it seemed a most fitting show place for the ensemble.

Dr. Latta's account of the cyclone which caused much destruction and the loss of three lives at Wagon Mound was most interesting. Most of the members had passed through Wagon Mound en route only a day or two after the hard storm and had a view of the wreckage.

Much credit is due the members of the Colfax County Medical Society for the splendid program of scientific papers and for the measures taken for social enjoyment.

"On to El Paso" was the slogan adopted, to the big Southwestern Meeting to be held there in November, 1930. Many of those present at this meeting announced the intention of attending the sessions and the clinics which the El Paso members assert will be the best ever.

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MEDICAL & SURGICAL ASSOCIATION OF THE SOUTHWEST

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DR. W. WARNER WATKINS ..... Phoenix

SOUTHWESTERN MEETING TAKING SHAPE

Never before in the history of the Medical & Surgical Association of the Southwest have the plans for the annual meeting been so well formulated this far in advance. The Program Committee, under the leadership of the president, Dr. E. Payne Palmer of Phoenix, has planned a Clinical Conference superior even to the well-remembered one which Drs. Crouse and Waite gathered together three years ago.

Packed into the three days of this meeting (November 6, 7 and 8) will be a series of papers, addresses and clinics which will fill every hour of them. The complete program will appear in our September issue, but some of the main features will be the following:

Dr. Alton Ochsner, Professor of Surgery, Tulane University, New Orleans, will discuss the subject of "Acute Craniocerebral Injuries."

Dr. Russell A. Hibbs, Professor of Orthopedic Surgery, Columbia University College of Physicians and Surgeons, New York, will present the subject of "Joint Tuberculosis."

Dr. George R. Herrman, Assistant Professor of Medicine, Tulane University, New Orleans, will present the subject of Cardiology, with two illustrated lectures; one "The Diagnostic Criteria of Heart Disease," and the other "The Treatment of Syphilitic Aortic Disease in the Last Stages."

Dr. Joseph Brenneman, of the Children's Memorial Hospital, Chicago, will represent the subject of Pediatrics; the title of his address has not yet been received.

Dr. William M. Spitzer, of Denver, Colo., will present the subject of Urology, giving an address and holding clinics.

Dr. Walter C. Alvarez, of the Mayo Clinic, Rochester, will represent the subject of Gastro-Enterology, giving a talk on "Practical Points in the Treatment of Gastro-Intestinal Disease." He will also give a dry clinic on this subject, using lantern slides or motion pictures.

Dr. Philip H. Kreuscher, of Chicago, Professor of Clinical Surgery, Loyola University, will present the subject of General Surgery in two addresses; one on "Fractures Near Major Joints" and the other on "Chronic Osteomyelitis."

Dr. M. Edward Davis, Instructor in Obstetrics at Northwestern University Medical School, Chicago, associated with Dr. DeLee, will present the subject of Obstetrics in a paper and in motion pictures.

Dr. James M. Martin, of Dallas, Texas, Professor of Radiology at Baylor University, will present the subject of Radiotherapy.

Dr. Roy Thomas, of Los Angeles, in charge of the Pneumonia Service at the Los Angeles General Hospital, will present the subject of the "Management of Lobar Pneumonia."

Dr. Charles T. Sturgeon, of Los Angeles, on the Surgical Service of the Los Angeles General Hospital, will present a paper on "The Use of Sodium Amytal in Surgery."

Dr. Allan K. Krause, of Tucson, Arizona, will give an address, the title of which has not yet been receied.

Dr. Robert Stanley Flinn, of Prescott, will represent the district of Arizona on the program, giving a discussion of "Diabetes."

Dr. Leroy S. Peters of Albuquerque, will represent New Mexico on the program, his paper being on "Cauterization of Adhesions" —Jacobaeus-Unverricht Method."

Dr. Jim Camp of Pecos, Texas, has been asked to represent western Texas on the program.

Dr. Carl Jumper of Torreon has been invited to represent northern Mexico.

Dr. Edward Jackson of Denver, has been invited to represent the subject of Ophthalmology.

There may be one or two additions to the program, but the above will justify the claim that this Conference will be a very attractive meeting.

The El Paso County Medical Society will prepare clinics to occupy all available time not taken up by lectures and papers.

There will be sufficient entertainment to break the monotony of the meeting, without interfering with its main objects,—to present a high class scientific program and postgraduate Clinical Conference for the medical profession of the Southwest.

SAMUEL BROWN NORRIS

Information comes to us of the death on May 27th of Dr. Samuel B. Norris of Tucson, a member of the Pima County Medical Society, and Fellow of the American Medical Association.

Dr. Brown was a graduate of the University of Illinois College of Medicine, class of 1908, and was forty-four years of age. The cause of death is said to have been pneumonia.

ROBERT CRAIG DRYDEN

The sudden death, in an automobile accident, of Dr. R. C. Dryden, of Pima, Ariz., removed one of the few remaining pioneer physicians of Arizona. Dr. Dryden was one of the charter members of the Arizona State Medical Association, being a young Santa Fe surgeon at Winslow in 1892 when the Association was organized. He was born in 1857, and was seventy-three years of age. He graduated in 1880 from the Missouri Medical College, which school merged with Washington University in 1899. As stated above, he was located at Winslow during the early years of his work in Arizona. For several years he has been practicing in Graham County, being instrumental in helping to organize the Graham County Medical Society two years ago. He was the delegate from that society to the Arizona State Medical Association in Phoenix this year, and it was difficult for those who did not know otherwise, to believe that the little doctor with the black hair and sprightly frame was more than seventy years old. When killed he was transporting some Boy Scouts to their camp in the White Mountains. He left no immediate family, being a widower with no children.

HOSPITAL CONSTRUCTION IN ARIZONA

Work has been started on the new wing to the Good Samaritan Hospital in Phoenix, which will add sixty more beds to the capacity of the institution, as well as providing some other greatly needed facilities. A new power plant will be built in a separate building; this will include a laundry. Among the features of special interest to the medical profession will be an auditorium which will seat about 250 people. This will be used primarily for the Nurses' Training School, but doubtless will be used much for medical gatherings, as well.

The new Southern Pacific Hospital in Tucson has also been started, and will be pushed to early completion. This will be a notable addition to the institutions for treating the sick in Arizona. Most of the work now being sent to San Francisco, out of Arizona, will be handled in the Tucson institution.

The Grunow Memorial Clinic Building has started construction in the block just west of the Good Samaritan Hospital. This building is designed to accommodate a number of doctors, with laboratories for clinical and x-ray work. It is being built by Mr. Grunow, of Chicago, as a memorial to his daughter.

DR. E. PAYNE PALMER, of Phoenix, has returned from the American Medical Association meeting in Detroit, where he was a member of the corps of demonstrators in the treatment of fractures. Enroute home he stopped in El Paso for conference with the Local Committee with regard to the forthcoming meeting of the Medical & Surgical Association of the Southwest, of which he is president.

DR. R. J. STROUD, of Tempe, State Superintendent of Public Health, has returned from attendance at a series of meetings, including the American Medical Association in Detroit, where he served as the delegate from Arizona.

DR. MAYO ROBB, of Phoenix, has returned to his practice from attending the American Medical Association meeting in Detroit.

DR. W. O. SWEEK, of Phoenix, has been visiting in Chicago during the first half of July.

DR. W. A. HOLT, of Globe, information reaches us, has been seriously ill for some weeks, with a generalized infection.

DR. T. W. WOODMAN and wife, of Phoenix, have returned after an absence of a month. They visited eastern clinics and attended the American Medical Association in Detroit.

DR. CHARLES B. PALMER, of Phoenix, has returned after a year's absence in postgraduate work in Paris, London and Boston. His work was chiefly in urology and urologic surgery, to which he expects to confine his practice in the future.

EL PASO COUNTY MEDICAL SOCIETY

Regular Meeting, May 12, 1930.

Meeting called to order at 8 p. m. by the President, Dr. Paul Gallagher. There were present 27 regular members, 13 honorary members, and one visitor (Col. Crimmons).

Minutes of the previous meeting were read and approved.

The first number of the evening was on Childhood Tuberculosis," by Dr. R. B. Homan, and consisted of lantern slides illustrating the various forms. Childhood type of tuberculosis, as defined by the American Sanatorium Association, is the term used to describe the diffuse or nodular lesions in the lungs and associated tracheo-bronchial nodes that result from a first infection of the pulmonary

tissue with the tubercle bacillus. McPhedran has classified the various forms according to lesions. These are viz:—

A. Focal tuberculosis, caseous or calcified nodules.

B. Tuberculous consolidation of a lobe or wedge, progressive or unstable.

C. Tuberculous consolidation of a lobe or wedge, retrogressive and benign.

D. Diffuse, childhood type, tuberculosis infiltration.

E. Miliary tuberculosis.

F. Tuberculosis of tracheobronchial glands, uncalcified.

F. Tuberculosis of tracheobronchial glands, calcified.

H. Apical, adult type infiltration of children and adolescents.

In type "A" theprognosis is good, only caseous unencapsulated focus in an infant is serious. In type "B" the prognosis is uncertain until this type "B", which is serious, is differentiated from "C." In type "C" the prognosis is good once it has been differentiated from "B." In "D" the prognosis is good unless the lesion increases in density; if so, it is serions. In type 'E" recovery is extremely rare. In "F" prognosis is very serious if multiple. In this there is danger of miliary tuberculosis or caseous pneumonia. 'G", warranting oversight from 10th to 20th year because often followed by apical lesion, especially if contagion is recent or prolonged. The prognosis in "H" is always precarious.

Dr. Waite then showed specimens from two infants who had died of tuberculosis. The first was the miliary form and was from a child two and one-half years old. On first glance the child seemed well nourished but on further examination it was seen that the tissues were full of fluid and that there was very little muscular tissue left, the child being really quite emaciated. We see a great number of cases of this type among Mexican children. It is usually hard to diagnose miliary tuberculosis in an infant and a history of exposure is quite important. Usually at autopsy a large solitary tracheobronchial gland is found, which is very caseous and breaking down. In the case present, however, this was not evident; instead, caseous mesenteric glands were found.

The second specimen was that from an infant 32 months old who had had an acute illness of twenty days duration. This correspoded to type "B" wherein whole lobes were consolidated. It was really a tubercular form of lobar pneumonia.

Dr. Waite stated that glands do not cast shadows in the x-ray unless they are extremely large or calcified. You can not make the diagnosis from the film alone.

The second number on the program was a paper entitled "Urethroscopy of the Pathologic Posterior Urethra" by **Dr. S. Elliot Wilson.**

Although only one and one-half to two inches in length, the posterior urethra is subject to many pathological derangements. This is especially true of the verumontanum which should always be closely studied in any examinatioin of the posterior urethra. The anesthetic of choice is a solution containing ½% cocaine hydrochloride, ½% sodium bicarbonate, and ¼% chloretone. Immediately before use two drops of adrenalin chloride (1-1000) are added to each drachm. Four drachms are used in all, one drachm being injected every three minutes and being massaged back into the posterior urethra and bladder. The verumontanum is seen as a projection from the floor of the urethra on withdrawal of the urethroscope from the bladder into the posterior urethra. It is in the form of a dome or cone or some variation of either. It is enlarged if it almost completely fills the posterior urethra or touches the walls without any evidence of the lateral sulci. Pathological changes encountered are simple congestion, atrophy, hypertrophy, deformities of all types, polypi or papilloma, and granulomata. In seminal vesiculitis the veru should be eliminated and treated thoroughly. In chronic prostatitis, the prostatic follicles are quite often red and swollen. Fulguration and local applications are used in treatment. Fulguration through the urethroscope in certain types of prostatic obstruction has a distinct place in the treatment of these disorders. Many cases of obstruction of the urethra will be found to be due to enormous hypertrophy of the veru, tumors of the veru, congenital folds or valves or stones. Tumors are generally papilloma or polypi and granulomata and are often the cause of a persistent urethral discharge due to constant irritation and stimulation of the adjoining glands. They are also the source of many sexual disturbances and irritations. They are easily destroyed by fulguration. Every case of partial obstruction in boys should be thoroughly examined for stones or congenital folds. Stones if not too large can be removed very easily through the urethroscope. The veru is found either atrophied or hypertrophied in sexual irritation, impotence, and other sexual disturbances. Some men claim that a masturbator can be told by the appearance of the veru. Hematuria, especially terminal, is often due to granulations in the post-montane space or to a very vascular prostate. So all cases should therefore have an examination of the posterior urethra if no cause can be found in the kidneys, ureter or bladder. Diverticuli and ectopic ureters can only be discovered by such an examination. No cystoscopic examination should be considered complete without a careful study of the posterior urethra, and with the cystourethroscope, it can be done in a single examination.

Dr. Jamieson: Dr. Wilson has considered the subject so amply that there is hardly anything left to say. I am just a little afraid of cocaine, especially after my experience with butyn which is supposed to be much less toxic. I use 20 c.c. of a 2% novocain sol. Have seen many cases complaining of pain on intercourse, too rapid ejaculation, and in the terminal stage, impotence. Practically all of these cases show a congestion of the whole posterior urethra; the verumontanum presents a juicy appearance and bleeds on the slightest touch. The application of a 10% sol. of silver nitrate to these cases usually suffices to effect a cure. Fulguration also is useful, but as a rule it is difficult to obtain sufficient anesthesia to stand the spark, which is quite painful.

Dr. Multhauf: This is a devilish good paper. The routine examination of the posterior urethra after an acute gonorrhea is too often neglected. There is bound to be some residual inflammation there and unless treated properly these cases will come back later on. The too frequent application of silver nitrate to the verumontanum is to be condemned, as it will have a deleterious effect on this delicate structure and will eventually cause atrophy.

Dr. K. D. Lynch: I want to congratulate Dr. Wilson on his paper. As to the anesthetic used, undoubtedly cocaine is toxic, and if used over a long service you will eventually get unfavorable reaction. This is true of any anesthetic, however, regardless of how low its toxicity. Dr. Jamieson's unfortunate experience with butyn probably would not be repeated again, in thousands of cases. A very careful examination should always be made of the anterior and posterior urethras with the urethro-

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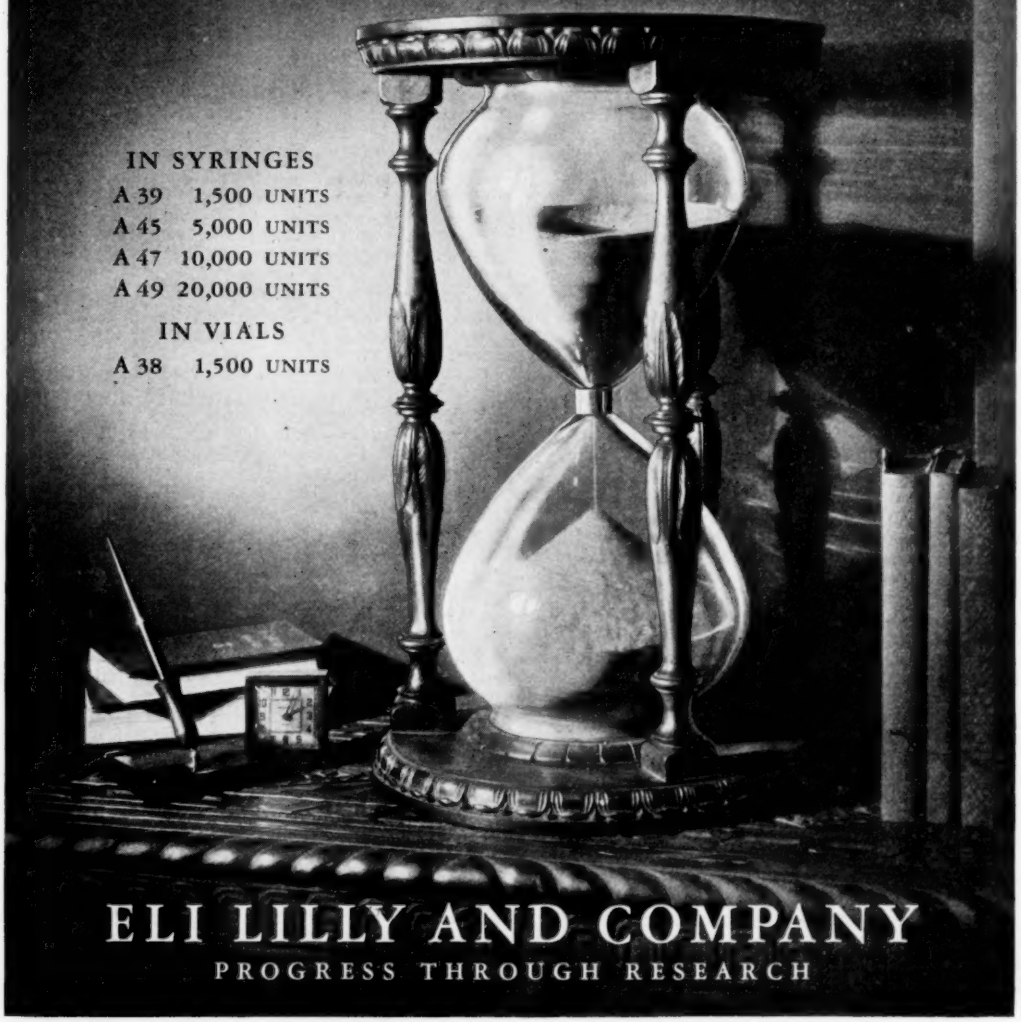

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PROGRESS THROUGH RESEARCH

scope, but I think that there is a lot of meddlesome work being done here by incompetent men. These men often make repeated applications of silver nitrate from which the patient never recovers. Repeated urethroscopic treatments are often meddlesome and cause more trouble than good.

Major Soper: There is only one procedure here that gives a really good anesthesia and that is caudal block. I have seen them all and have found blocking hte most satisfactory.

Col. M. L. Crimmens, U. S. A., then gave the society a brief sketch of the progress that has been made in recent years in the treatment of snake bites. He stated that he and his co-workers have analyzed in the past three years over one thousand reports of snake-bites. He found that the deaths were due to delay in instituting proper treatment. In the majority of cases potassium permanganate had been used and when it was found inadaquate, the patients were then sent to him for antivenom treatment. Potassium permanganate plays no part in present day treatment. Mechanical suction in conjunction with anti-venom is the treatment of choice. But the mechanical suction must be kept up till results are obtained—often for twelve to fifteen hours. Never discontinue it simply because you have given anti-venom. The action of the venom is similar, in end results, to that of hemorrhage and so it is necessary to keep up the volume of the blood. Saline is given and when obtainable, blood intraperitoneally or intramuscularly if the case is not urgent, transfusion into the vein for quick results, when available. A blood count should be made every three hours and when the R. B. C. and H. b. get too low antivenom is given every hour or so. Mechanical solution is best accomplished by means of a little rubber bulb similar to a breast pump. A rubber tourniquet is applied at once above the bite. The latter is laid open by an "H" incision, whose limbs are two inches long one-fourth inch deep. Fangs are sought for and removed when found as they sometimes carry tetanus bacilli and gas bacilli. Suction is then applied. The limb will swell. When this swelling increases to twice the natural size a bracelet of cross-incisions is made just distal to the tourniquet; the latter is removed and placed at a higher point, etc. Hot compresses of magnesium sulphate are useful in increasing the discharge from the wounds. 25 mgm. of rattle-snake venom is the minimum lethal dose. The average Texan rattler holds about 220 mgm. while at the Government institute as much as 1000 mgm. have been obtained by milking. It has been stated that in Brazil the average is only about 90 mgm.

After Col. Crimmen's talk Mr. Dumont of the Petrolagar Laboratories, gave a moving picture illustrating the anatomy of the anterior abdominal wall. The dissection had been done by Howard B. Kellog. A thousand feet of very instructive dissection was presented.

The president then read a communication from the secretary of the Texas State Board of Medical Examiners calling attention to the large number of itinerant fakers that are traveling over the state visiting the larger cities and giving lectures on so-called "new" discoveries in diet and methods of diagnosis, etc. He urged that vigorous action be taken against them and that his office be advised of the results.

The president announced that Dr. Hardy was sick in Masonic Hospital and asked the members to visit him.

There being no further business the meeting adjourned at 9:30 p. m.

S. H. NEWMAN,
Secretary.

EL PASO COUNTY MEDICAL SOCIETY
Meeting of May 26, 1930

Meeting called to order at 8 p.m., by the president Dr. Paul Gallagher. There were present 39 regular members, 19 honorary members and 3 visitors, Dr. Camp, of Pecos, Dr. Gregory and Dr. Rasmus (D.D.S.).

The minutes of the previous meeting were read and approved.

The president read a communication from the University of Bordeaux, France, announcing a postgraduate course for this summer in ear, nose and throat work. Another announcement was read concerning the semi-annual clinic of the Tarrant County Medical Society starting June 19th. A letter was then read from the Legislative Committee of the State Medical Association urging that immediate action be taken in an endeavor to get Senators and Representatives in the Texas Legislature who are favorable to legitimate medicine.

Dr. Swope then reported the following clinical case:

American, age 30, public accountant. Past history irrelevant. In 1925, he had an apparent spontaneous collapse of the right lung; in 1927, a collapse of the left lung, and in February this year, another collapse of the right lung. All of these collapses followed close upon a severe nervous strain and were all entirely relieved within thirty days. Following the second collapse in 1927, he was examined by Dr. Werley and Dr. Egbert and an x-ray examinatioin was made by Drs. Cathcart and Mason. All concurred in a diagnosis of spontaneous pneumothorax. An examination now shows an apparently normal lung and the x-ray fails to reveal any pathology. The patient's chief comphlaint is nervousness, inability for continuous application, irritability, insomnia and sweating of hands and feet. Physical examination, entirely negative. Dr. Swope doubted seriously if actual collapse ever actually occurred in spite of the x-ray findings in 1927, which showed the pleural space filled with air.

Dr. W. W. Britton, who examined the patient together with Dr. Swope and who made x-rays recently, stated that this was a most peculiar care and he was convinced that it was one of mistaken diagnosis. There is no pathology now, there had been no loss in weight, no fever and no hemorrhage. It does not seem possible for such a condition to have existed with such complete recovery in such a short length of time without treatment of any kind. The x-ray of 1927 undoubtedly shows collapse of about the outer third or fourth of the lung without adhesions. The x-ray now is perfectly normal. It made him think of a case sent here from Dallas two years ago to Dr. Ralph Homan, which went on to autopsy. There Dr. Turner found he had a bullous emphysema. So he thought likely that was the true diagnosis in Dr. Swope's case.

Dr. Ralph Homan stated that the case referred to by Dr. Brtiton had been sent to him with a diagnosis of bilateral pneumothorax and that the immediate cause of death had been coronary thrombosis.

Dr. Waite said he wondered if it was not harder to get over a case of bullous emphysema than one of pneumothorax.

Dr. Laws said that in post-mortems we often see small areas of emphysema near the surface of the lungs. He thought that this case was not one of pneumothorax but one wherein an emphysematous bleb had ruptured into the pleural cavity.

Dr. Ralph Homan thought it was pretty hard to figure out how, with an emphysematous bleb rupturing into the pleural cavity, the patient could be up and about apparently well within thirty days.

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The Mead Policy that for years has proved itself professionally and economically valuable to physicians who feed infants also applies to Mead's Viosterol in Oil, 100 D (originally Acterol). As with Dextri-Maltose, we feel that Mead's Viosterol is a part of the physician's armamentarium, to be prescribed by him alone. Therefore, we refrain from lay advertising of Mead's Viosterol or any other Mead Product; furthermore, we do not print dosage directions on the bottle, on the carton or in a circular. "Hold fast to that which is good" —the Mead Policy, Dextri-Maltose and Mead's Viosterol (originally called Acterol).

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MEAD'S INFANT DIET MATERIALS ARE ADVERTISED ONLY TO PHYSICIANS. NO FEEDING DIRECTIONS ACCOMPANY TRADE PACKAGES. INFORMATION IN REGARD TO FEEDING IS SUPPLIED TO THE MOTHER BY WRITTEN INSTRUCTIONS FROM HER DOCTOR WHO CHANGES THE FEEDINGS FROM TIME TO TIME TO MEET THE NUTRITIONAL REQUIREMENTS OF THE GROWING INFANT. LITERATURE IS FURNISHED ONLY TO PHYSICIANS

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Dr. Cathcart could not agree that this was a case of bullous emphysema. The x-ray taken in 1927 shows a uniform filling of the pleura with air.

The next number on the program was the showing by lantern slides of "Some Interesting Dermatological Conditions," by **Dr. Leslie M. Smith.** The views presented were, viz:

1. Case of epidermolysis bullosa.
2. Case of congenital hyperkeratosis of palms and soles.
3. Cutaneous horn.
4. Lichen planus, generalized.
5. Erythema induratum.
6. Scrofuloderma.
7. Tuberculosis verrucosa cutis.
8. Blastomycosis of leg.
9. Mycetoma.
10. Dermatitis gangrenosa.
11. Early case of leprosy, maculo-anesthetic type.
12. Nodular type of leprosy.
13. Case of rupial secondary syphilis.
14. Ringworm of nails and feet.
15. Bromoderma.

The last number on the program was the presentation of motion pictures illustrating the 'Anatomy of the Abdominal Viscera." It was presented through the courtesy of the Petrolagar Laboratories.

Under unfinished business, Dr. Waite outlined plans for the meeting of the Medical & Surgical Association of the Southwest to be held hear next Nove greatember. The Executive Committee was showing great enthusiasm in putting on the best meeting held so far. A number of distinguished guests had already been booked. It was planned to advertise the meeting extensively and a large attendance was anticipated. A five-dollar registration fee would probably take care of all the expense.

Under miscellaneous business, Dr. Outlaw stated that the Health Department was getting cooperation from only about 50% of the physicians in reporting contagious diseases. The Medical Officers of Fort Bliss are reporting 100% the cases of contagion among their school children. He was especially anxious to have the doctors report their tuberculous patients, mainly for statistical purposes. To this end he proposed to get out post cards and distribute them to the doctors on which they would be given more of our support and thought that if a to get the full cooperation of the Society in reporting all diseases made reportable by law.

Dr. Laws felt that the Health Department should be given more of our support and thought thta if a little pressure were brought to bear, we would probably do better. In regard to reporting tuberculosis, however, some men would probably object. He recalled that several years ago the State Health Officer sent out requests for such reports. The patients whose names were sent in were immediately flooded with literature. In many cases this was resented by the patient. As Councilor of the State Association, he had been asked to come before the County Society and request that all doctors be punctual in reporting their births, so that Texas might be admitted to the Registration area.

Dr. Waite suggested that the proposed post cards for reporting tuberculosis might include questions, the answering of which would indicate whether or not the case should receive further attention from the Health Department.

Dr. Outlaw said that these reports would be strictly confidential and he promised that no literature of any kind would be sent the patient. One purpose of these reports was the curtailment of unauthorized rooming-houses and practitioners from preying on these people.

Dr. Waite then made a motion that Dr. Outlaw enforce the law dealing with the reporting of contagious diseases. Motion seconded and carried.

Dr. Frederick H. Blanchard's application for membership was then read. On motion by Dr. Barrett, seconded by Dr. Laws, he was unanimously admitted into the Society.

Dr. Gallagher announced that Dr. Hardy, who has been sick at Masonic Hospital, was very much improved.

The President then announced the following incomplete committee personnel for the Southwestern Association meeting:

Advisory Committee: All past-presidents of the El Paso County Medical Society.

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No Starch
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Strictly starch-free, palatable muffins, bread, cakes, pastry, etc., are easily made in any home from Listers Flour. Recipes are easy to follow and Listers Flour is self-rising. One month's supply $4.85
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3. **Special Courses:** Orthopedica and Traumatic surgery; gynecology and radiation therapy; eye, ear, nose and throat, thoracic, genito-urinary and goiter surgery; bronchoscopy, etc.

All courses continuous throughout the year. Detailed information furnished on request.

Executive Committee: Dr. Paul Gallagher, Dr. J. W. Laws, Dr. S. H. Newman, El Paso; Dr. W. Warner Watkins, for Arizona.

Banquet and Entertainment: Dr. W. R. Jamieson, Dr. James J. Gorman, Dr. John E. Morrison.

Hotels and Ralroads: Dr. R. L. Ramey, Dr. F. P. Miller, Dr. C. M. Hendricks, Dr. J. H. Gambrell.

Guests: Dr. K. D. Lynch, Dr. H. E. Rogers, Dr. A. W. Multhauf, Dr. F. C. Goodwin.

Golf: Dr. James Vance, Dr. Ralph H. Homan.

Scientifice Exhibits: Dr. G. Werley, Dr. John W. Cathcart, Dr. E. A. Duncan, Dr. George Turner, Dr. J. Mott Rawlings.

Round Table Luncheons: Dr. W. L. Brown.

Lantern Slides: Dr. A. P. Black.

Commercial Exhibits: Dr. T. J. McCamant, Dr. Paul E. McChesney.

Hospital Clinics:
Masonic—Dr. F. P. Miller
Providence—Dr. Frank P. Schuster.
Hotel Dieu—Dr. E. W. Rheinheimer.
City-County—Dr. S. H. Newman.

Reception—Hotels and Trains: Dr. R. B. Homan, Dr. S. A. Schuster, Dr. J. A. Pickett, Dr. Harry Leigh.

Transportatoin: Dr. O. E. Egbert.

Dr. Egbert then gave the report of the Radio Committee covering the six months' radio program that had been voted by the Society late last year. The program has now been completed with the exception that we still owe some money to the broadcasting stations. Fifty-nine members have donated $6.00 each, but there is still owing $153.50. This makes the entire six months' program to have cost $507.50. He wanted to know how the Society proposed to take care of the deficit.

The Chair thought that this deficit should be made up by voluntary contributions and should not come out of the Society's funds. He called for a volunteer Committee to ask for contributions from the members. Dr. Waite, Dr. Rlaph Homan and Dr. Outlaw volunteered to serve on such a committee.

Dr. Gorman then called the Society's attention to the fact that most of the papers presented at our meetings are not turned in and consequently we are not getting our share of space in Southwestern Medicine. He offered to have the papers typewritten if the members would only turn them in in proper shape.

The president then called the Society's attention to the recent death of our honorary member, Dr. Howard Thompson, and appointed Dr. Swope, Dr. Werley and Dr. J. A. Rawlings, as a Committee to drawn up resolutions expressive of the Society's sentiment.

There being no further business, the meeting adjourned at 10 p. m.

S. H. NEWMAN, M. D.,
Secretary-Treasurer.

Founded 1896 by Dr. Hubert Work

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New Equipment

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Superintendent

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For Nervous and Mental Disorders and Allied Conditions
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Approved diagnostic and therapeutic methods used.

Occupational therapy, recreation and entertainment.

G. Wilse Robinson, M. D., Medical Director

G. Wise Robinson Jr., M. D., Association Med. Dir.
Paul A. Johnson, M.D., Resident Physcian and Internist.

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Kansas City, Missouri

MINUTES OF THE MEETING OF THE THIRTY-NINTH ANNUAL SESSION OF THE ARIZONA STATE MEDICAL ASSOCIATION, HELD IN PHOENIX, APRIL 24 TO 26, 1930.

(Continued from June Issue)

REPORT OF THE COMMITTEE ON MEDICAL DEFENSE

Your committee on medical defense has taken official cognizance of five separate cases during the year. They were the Looney case (Prescott); Perkins-Gungle-Langham case (Morenci); Tarasoff, (Phoenix), and the Knotts case (Yuma). In the Morris Hospital case in Safford we acted only in an advisory capacity.

The Looney case was refused a new trial; the Langham case was non-suited; the Tarasoff case was non-suited. The Knotts case was an expensive bitterly contested situation.

At the present writing we have but one threatened case for consideration.

Defense Fund Receipts	$ 4,802.75
Expenses	1,699.15
Total amount available for Med. Defense	12,816.04

During the current year we have expended $2216.53; of this amount $1485.63 was used to defray the expenses of the Knotts case in Yuma. With a membership of ten at $6.00 each for medical defense it would take Yuma County 25 years to offset this expense, providing they do not have any further difficulty. The total sum expended in Yuma county during the period of existence of our Medical Defense has been $1642.63; at the same basis it will take Yuma County 27 years to reimburse the State Association.

This situation is cited to illustrate how expensive it is for physicians to criticize one another.

J. E. BACON,
Chairman Med. Defense Committee.

EXPENSE OF MEDICAL DEFENSE SINCE ITS INCEPTION IN 1916

11-21-17—John W. Flynn	$ 519.45
7-23-18—Kaull case	50.00
6- 6-19—E. Payne Palmer	114.80
5- 5-20—O. H. Brown	50.00
6- 3-20—Shackelford - Nelson	43.65
7-23-18—Bim Smith	552.35
10-20-20—Tindall case	27.00
5- 5-20—Wilkinson	2203.05
1-14-22—Smelker	365.93
1-21-27—Melick	145.45
9-25-28—Looney	388.35
5- 3-29—Laugharn - Gungle	155.90
5- 3-29—Knotts	1485.63
7- 9-29—Tarasoff	150.00
3-22-26—Campbell	130.00
Total	6381.56

COMMENT BY ASSOCIATION ATTORNEYS

Dr. D. F. Harbridge,
Secretary, Arizona State
Medical Association,
Phoenix, Arizona.

Dear Sir: During the past year our previous record of no recoveries in mal practice cases has been maintained. During that time we have had only two or three new cases, one of the cases the case of Mammon vs. Drs. Laughran and others in Greenlee County is still pending. It was set for trial early in the winter, but about the time of the setting the attorney for the plaintiff requested that the setting order be vacated, and stated that in his opinion he would not be able to establish a case, and that case will in all probability be dismissed without further proceedings.

In the case of Chris Christainsen vs. Drs. Looney and Allen of Prescott, the judgment of the Superior Court of Yavapai county was affirmed by the Supreme court of this state. In that case the law, by the opinion of the court, is now established in this state with the following effect that no civil liability of any kind or character whatever may attach to any physician acting as a member of the Sanity Board appointed by any court of this state, unless it can be affirmatively shown that the judgment or decision of the physicians as to the sanity of the defendant was actuated by malice or fraud. In other words, the opinion and judgment of a physician acting as the representative of the court in the determination of the question of insanity of any defendant, even though the conclusion reached by the physician be erroneous, does not make him liable civilly.

In the case of Mowery vs. Tarasoff tried in the Superior court of Maricopa county, Arizona, the court at the close of plaintiff's case, directed a verdict in favor of the defendant and against the plaintiffs, and dismissed the case as against Dr. Tarasoff upon the ground that there was no medical testimony substantiating the charges of malpractice contained in the complaint.

A situation of interest to the Association has arisen in Safford, Arizona. Two physicians there have erected a modern hospital suitable for the handling of all cases originating to that county. This hospital is placed upon a lot in the residence district. The owner of an adjoining lot brought an action to enjoin the erection and conduct of that hospital upon the ground that it was a menace to the health, safety and comfort of the surrounding inhabitants of the town. The case has not yet been tried, but from the preliminary attacks upon the complaint, the court has taken the view that the modern hospital is not in itself a nuisance and that it is not a menace to the health, safety or comfort of the owners of adjoining property or the residents of the surrounding neighborhood. This case is not one which is being defended by the Medical Defense Committee of the Association, but it is one of general interest to physicians. We believe that the Superior Court will not sustain the injunction and that such will be the ruling of the Supreme Court of this State if it is carried that far. Unless there are some restrictions running with the land upon which the hospital is built or a zoning ordinance in effect in the city where located, and unless it can be shown that the hospital is being conducted in such a way as to be unsanitary and not in accordance with modern methods, we believe that a hospital can be erected anywhere in a city.

A large part of the success in the treatment of these mal practice cases must be attributed to the splendid cooperation of the members of the Association in rendering every aid possible in the defense of its members. Without this hearty co-operation we could not have hoped to attain the success which we have with these cases, and the importance of this feature of the work cannot be over-emphasized.

We wish further to say that we believe that the freedom of the members of this association from suits of mal practice can be in no small measure attributed to the high standard of ethics and rigid requirements as to education and training required by members of this association before physicians are licensed to practice medicine in this state and permitted to become members of this Association, and such freedom from such suits can

only be maintained by rigidly adhering to the high standard adopted by the members of your profession.

Trusting that the members of the Association will continue to be free from the annoyance of such suits, and thanking the members of your Association for the assistance rendered us during the past year, we beg to remain

Respectfully yours,
SLOAN, HOLTON,
McKESSON & SCOTT,
By CHARLES R. HOLTON.

REPORT OF THE COMMITTEE ON NATIONAL LEGISLATION, ARIZONA STATE MEDICAL SOCIETY

R. J. Stroud, M. D.

At present there are several Bills before Congress relative to various phases of national legislation which affect doctors. Among them are:

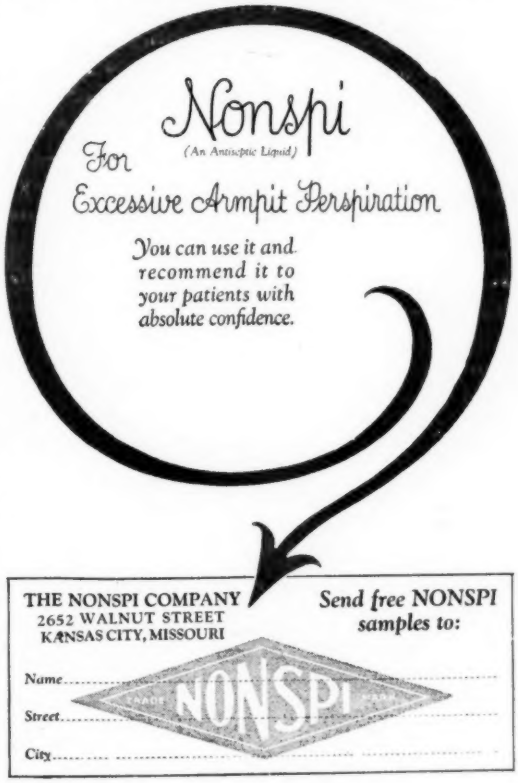
1. Federal Control of Narcotic Drugs.
2. Federal Control of the activities of the states in the field by hygiene of mothers and children.
3. Provision at the expense of the Federal Government of medical surgical nursing, and hospital service for rich and poor veterans suffering from diseases and injuries having no relation to military service.

No. 1.Federal Control of Narcotic Drugs. Representative Porter has drawn up several Bills relating to narcotics—Bills 3053; 9054; 10561 and 11143. The Bill 10561 is the best of the lot because Representative Porter was approached by the representative of the American Medical Association to see that the rights of the physicians were upheld. All of these laws do the physician no good and do a great deal of harm. They do not propose to have any cooperation between the states and Federal Narcotic Division and the physician would be licensed by a veritable autocrat in the Commissioner of Narcotics and we would be subject to his whim. The charge that a great many physicians are themselves addicted to the use of narcotics has never been substantiated by figures and the narcotic division claims that physicians can get themselves large amounts of morphine for their own addiction. The American Medical Association claims that this is not true. In Bill H. R. 11143 there is no provision for the scientific research of rare alkaloids of narcotic drugs while it allows certain importers to import coca leaves which do not contain cocaine or ecgonine in any quantity. The chief objection to all of these Bills is to substitute a single federal officer ranking as a commissioner in place of a Federal Narcotic Control Board made up of three cabinet officers. There is not time to go into the other features of the Bill.

No. 2. Federal Control of the activities of the states in the field of hygiene of mothers and children. There have been many ilbls which can be called miscellaneous Sheppard-Towner Bills. These are other proposals to substitute state control with federal domination. As a matter of fact the states whom they help finally have to pay the taxes, the state taxes being paid into the Federal Treasury. The Bill which more nearly proposed something good is the Cooper Maternity and Infancy Health United Bill H. R. 9888. The State of Arizona raised its quota to double at the last session of the legislature to meet the loss of federal funds because the Sheppard-Towner Act was to be suspended by agreement and by the same token any other state could supply those needs. The trouble with a legislation of this kind is that it places lay people in charge of these monies and they have professional people working under them. The duties they have is to see how many nice forms can be submitted to the Federal Government. H. R. 9888 provides for $1,500,000 to be expended—half to the Welfare of Mothers and Children and the other half to the local health units or organizations. Of the half turned over to the Welfare of Mothers and Children there is no known way of administering this in the State of Arizona except through the Child Welfare Division which cannot use this money beneficially but federal subsidies of any other nature cannot go through because the women's organizations throughout the country want children welfare bureaus sustained. Under the other provision of the Bill the $750,000 can be expended wisely through the state health departments in different counties because it is entirely under the control of medical men. Even at that provision is not made to use the whole appropriation. With all its defects a state with the comparatively limited wealth and rural tendencies as Arizona is, would get in federal subsidy a good many more dollars back when compared with a state as large as California. This is a better Bill than the Sheppard-Towner Bill because one-half of the monies are to be expended by medical health men directly. As a public health man, I can see where Arizona would be benefited by this Bill but the other Bills along this line would simply be a waste of public monies.

No. 3. The proovisions of medical surgical nursing and hospital service for rich and poor veterans suffering from diseases and injuries having no relation to military service. This presents a serious situation.

A report of the directors of the Veterans Bureau shows that on June 30, 1929, patients entering hospitals for non-service conditions and remaining un-



der treatment from that time, formed 43 per cent of the hospitalization. 27,437 were patients in the hospital of whom 11,823 were patients who were there because of diseases and injuries not connected with military service. During the fiscal year ending June, 1929, admissions to all hospitals for the treatment of non-service connected conditions numbered 56,892 or 58 per cent of all admissions. Admissions for service connected injuries decreased less than 1200 while those for non-service injuries increased nearly 12,000. Even with as much as 40 per cent of all hospital beds under control of the Veterans Bureau occupied by patients suffering from non-service connected injuries, there were on December 1st, 1929, only 184 veterans awaiting hospitalization for diseases and injuries connected with the service. Obviously these veterans could easily have been accommodated by ousting some of the veterans who were receiving treatment for non-service connected causes.

Moreover at that time money had already been appropriated, as yet unexpended, sufficient to provide 3100 additional beds. Notwithstanding this, the congress appropriated $15,950,000 for additional hospital construction. The conclusion is that this entire appropriatioin was for the treatment of veterans suffering from diseases and injuries non-service connected. You can figure out the approximate number of new beds that this appropriation provided and the cost per day for the Federal Government for maintenance besides the 3100 provided for under a previous appropraition. It is not unfair to state that these beds will only be needed for a relatively limited length of time to serve men and women who now rank as veterans, the war ended in 1918 and certainly there should be a constant decrease in the number of such persons living. Even if we allow for possible increasing illness due to advancing age, the time must come, and relatively shortly, when the number of possible patients will be very much reduced.

An interesting question would be, What will be done with the millions worth of investments in the hospital plants in the Veterans Bureau at that time. No one can begrudge veterans, suffering from diseases and injuries incurred in the line of duty, the money tha tthe congress can appropriate for them. In the interests of these men injured in the line of duty, the congress should conserve federal funds for their benefit—not spend them for the relief of disease and injuries not received in service, instead af putting 30 or 40 million dollars in hospital plants that will be of but temporary value and that will require enormous annual outlays for maintenance; and that after all will be available only to persons who can leave their homes for treatment; the congress would certainly have done much better to have provided for the rendering of medical and surgical service at government expense by local hospitals and physicians—or at least have provided adequate pensions to allow veterans who are disabled to meet their own wants with a respect not only to medical and surgical service but with respect to food, fuel and clothing.

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NERVOUS AND MENTAL DISEASES FOR NURSES: By Irving J. Sands, M. D., Associate in Neurology, Columbia University, N. Y.; Associate Neurologist, Montefiore Hospital, N. Y.; Attending Neurologist, Brooklyn Jewish and Brownsville Hospitals; Attendng Neuropsychatrist, United States Veterans Hospital, N. Y.; Illustrated; Philadelphia and London; W. B. Saunders Company; 1929.

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